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**Postexercise oxygen consumption and metabolic hormone
profile in lean and obese men**

by

Kristina Wong



A thesis submitted to the Faculty of Graduate Studies and Research in partial fulfillment
of the requirements for the degree of Master of Science

Faculty of Physical Education and Recreation

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University of Alberta

Faculty of Graduate Studies and Research

The undersigned certify that they have read, and recommend to the Faculty of Graduate Studies and Research for acceptance, a thesis entitled **Postexercise Oxygen Consumption and Metabolic Hormone Profile in Lean and Obese Men** submitted by **Kristina Wong** in partial fulfillment of the requirements for the degree of Master of Science



ABSTRACT

This study investigated the influence of body fat on energy expenditure following 30 minutes of cycling. Men aged 30-39 years ($\dot{V}O_{2\max}=48$ ml/ kg fat-free mass/min; body fat: obese $\geq 25\%$; lean $< 16\%$) participated. Variables measured on 2 occasions (Baseline and Exercise/EPOC) included $\dot{V}O_2$, tympanic temperature, heart rate (HR), blood lactate, respiratory exchange ratio (RER), cortisol, growth hormone (GH), thyroxine (T_4), and triiodothyronine (T_3). At rest, all variables were similar between groups except T_3 was higher in the obese vs. lean ($p<0.05$). In the lean group, lactate recovery coincided with the cessation of EPOC, while cortisol, RER, and GH recovery occurred later. In the obese group, lactate and GH recovery coincided with the end of EPOC, while cortisol and HR recovery occurred later. Following exercise, the obese, compared to lean subjects, consumed less oxygen, had higher RER and cortisol, and lower GH ($p<0.05$), which suggests blunted oxidative activity.

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List of Abbreviations

AT	Anaerobic threshold
AUC	Area under the curve
BMI	Body mass index
CNS	Central nervous system
EPOC	Excess postexercise oxygen consumption
FFA	Free fatty acid
FFM	Fat-free mass
GH	Growth hormone
GHRH	Growth hormone releasing hormone
HPLC	High performance liquid chromatography
LPL	Lipoprotein lipase
NE	Norepinephrine
PDH	Pyruvate dehydrogenase
RER	Respiratory exchange ration
RMR	Resting metabolic rate
SNS	Sympathetic nervous system
T ₃	Triiodothyronine
T ₄	Thyroxine
TEE	Total energy expenditure
TEF	Thermic effect of feeding
T _{1/2}	Half time for the rate of decay
TG-FA	Triglyceride-fatty acid
VE	Ventilation
V _{C02}	Volume of carbon dioxide expired

$\dot{V}O_2$	Volume of oxygen consumption
$\dot{V}O_{2\max}$	Maximal oxygen uptake
$\dot{V}O_{2\text{peak}}$	Peak oxygen uptake
V_T	Ventilatory threshold for $\dot{V}E/\dot{V}CO_2$

Chapter 1: Introduction

Overview

The present study was intended to investigate the relationship between body fat and energy expenditure, particularly during the recovery period from exercise. Excess postexercise oxygen consumption (EPOC) and hormones related to energy metabolism, and in particular fat metabolism, were the parameters of interest. The sample of participants was two groups of men ages 30 to 39 years distinguished by amount of body fat. Initial entry into the study was based on BMI (kg/m^2) in the ranges of 18.5 to 24.9 to represent a healthy weight group and 27.8 to 34.9 to represent an obese group. This classification is based on current interpretation of BMI described in health and medical literature (81).

Significance

Responsiveness of the metabolic energy system to changing demands is regulated differently in healthy weight versus obese populations. Differences in the mechanisms controlling energy balance are such that a permissive metabolic environment may favor a propensity toward obesity. Therefore, energy expenditure, comprised of resting metabolic rate (RMR), thermic effect of feeding (TEF), and thermic effect of activity (TEA), may be blunted in obesity. Accordingly, evidence of a depressed RMR (8,89) and TEF (58,121) has been reported in obese individuals. If this is true, then metabolic recovery from exercise, as a component of TEA, may also be blunted.

Upon the cessation of exercise, metabolic activity remains heightened for a transient period of time. The elevation in oxygen consumption is called “excess postexercise oxygen consumption” and is known as “EPOC”. The implication of a

prolonged EPOC is that the prescription of exercise aimed at maximizing EPOC may not be the same combination of intensity and duration as prescribed to maximize caloric expenditure during exercise. However, many authors have concluded that the energetic cost of EPOC is very small and of little significance for weight control (20,33,41,48,62,67). As well, there may be a threshold for intensity and/or duration of exercise before a prolonged EPOC is achieved and this may be greater than the capability of the average person exercising for weight control. Nonetheless, oxidative activity occurring during the recovery period from exercise provides important information into how energetic challenges are differentially regulated and may lend insight into why some people are susceptible to weight gain or resistant to weight loss.

Comparison of energy metabolism in healthy weight versus obese populations has shown varying biochemical and hormonal profiles at rest, in response to exercise, and during recovery from exercise. Increased metabolic efficiency in the form of low energy expenditure, a tissue enzymatic profile favoring lipid storage, altered neurohormonal control over metabolism (8,19), a reduction in nonconservative mitochondrial respiration, and decreased rate of triglycerol-fatty acid cycling (80) have been described in the propensity toward obesity. Control of these mechanisms of metabolic regulation is hypothesized to permit maintenance of normal body weight by dissipating excess energy in the form of heat. Obese subjects, therefore, may be observed to have a blunted response from the hormones that stimulate fuel mobilization and/or oxidation. Furthermore, it may be during recovery, rather than during exercise, that differences in how the body copes with changing energy demands and how it prioritizes metabolic fuel selection are most apparent.

Delimitations

The sample of participants examined was men ages 30 to 39 years. Entry into the study was based on BMI (kg/m^2) in the ranges of 18.5 to 24.9 to represent a normal weight group and 27.8 to 34.9 to represent an obese group. This classification was based on current interpretation of BMI described in health and medical literature (81). The (American) National Heart, Lung, and Blood Institute (NHBLI) obesity guidelines were developed by the institute using an “evidence-based model and methodology” that included a review of scientific literature found in MEDLINE from 1980 to 1997 (81). Evidence from approximately 394 randomized controlled trials (RTC) was considered by the institute’s panel of 115 health experts. Thirty-five key questions were identified and appropriate RTC articles were reviewed to address the issue and establish guidelines. Issues relating to BMI included: BMI to assess overweight and obesity; and BMI to establish relative risk of death and illness. The institute’s guidelines have been endorsed by various professional health societies (81). Subjects were excluded if metabolic conditions existed that would possibly confound the analysis of hormonal data. Exclusion criteria are listed in Chapter 3.

Limitations

Assumptions underlying the above mentioned measurements present limitations to the results of the study. The assumptions are as follows:

Aerobic Fitness

1. definition

There appears to be no widely accepted definition of fitness, nor any agreement as to what specific components should comprise a fitness evaluation. Fitness, for the

purpose of this study, was specifically the efficiency of energy utilization and related to cardiorespiratory fitness. Maximum oxygen uptake ($\dot{V}O_{2\max}$) has long been considered the “gold standard” for the evaluation of cardiorespiratory fitness (the maximal rate of energy production from oxidative processes). The value of specific fitness test items such as $\dot{V}O_{2\max}$ and the implications of data collected have been much debated.

2. measurement ($\dot{V}O_{2\max}$)

Technical and biological errors elevate inaccuracy of measurement. Technical errors involve the capability of the technician to run the test and collect data with precision and competence. Biological errors include variability in $\dot{V}O_{2\max}$ due to height, body size and composition, genetics, gender, age, and mechanical work efficiency (familiarity with the exercise mode). These variations are minimized when $\dot{V}O_2$ is expressed in terms of ml/kg/min (relative to body weight). There are also day-to-day variations within individuals due to nutrition and hydration status, prior exercise, and motivation. Since motivation plays a very important role in tests requiring maximal effort, standard criteria have been identified (specified on page 38 of Chapter 3) and were used in this study to denote achievement of maximum effort. Due to a greater degree of error in getting subjects naïve to exercise to perform maximally, the result is more precisely identified as “ $\dot{V}O_{2\text{peak}}$ ” rather than $\dot{V}O_{2\max}$. Nonetheless, $\dot{V}O_{2\max}$ remains the convention.

Diet record

Self-reported data are problematic in the reliability of what is reported. This type of instrument relies on the accuracy, honesty and consistency of the subject. As well intersubject differences are likely to exist. Standard procedures in the collection of data are specified on page 39 of Chapter 3.

Hydrostatic weighing

Hydrostatic weighing is an indirect assessment of body composition. There are technical and biological errors involved in computation of body fat. Technical errors involve the capability of the tester and accuracy of the equipment and biological errors involve the constants used in the Siri and Brozek equations (84) to assume the densities of fat-free mass (muscle, bone, viscera, water content, mineral content, lung volume, gastrointestinal volume, etc). These errors either overestimate or underestimate percent body fat.

Hydrostatic weighing also involves the assumption that the body is comprised of two homogenous compartments: fat and fat-free tissue (84). Adipose tissue is assumed to have a constant density of 0.90 and lean body mass of 1.10 (84). Densities of both compartments vary between people and within a person at different times. For instance, the hydration status of muscle can affect body fat determination by 0.4 to 4.0% (84).

Other sources of error include: water temperature (density of water at 35 degrees celsius is 1.0) and effort of the subject related to maximal exhalation in determining the residual volume of the lungs. Despite the potential sources of error, hydrostatic weighing has been shown to be highly reliable with a Pearson-product correlation coefficient greater than 0.95 (84). Standardized conditions of measurement that were used to minimize these variances included water temperature at 32 to 35 degrees celsius to avoid excess shivering and enhance subject comfort, minimal clothing worn by subjects to avoid trapping air, and subjects were asked to void their bladder and defecate if necessary prior to entering the tank (84). As recommended by Pollock and Wilmore (84), subjects were also asked to follow a normal diet, fluid intake, and exercise pattern the day prior to testing and to avoid foods that cause excessive amounts of gas in the gastrointestinal tract. As well, subjects were asked to abstain from eating or

smoking for a minimum of two hours prior to testing and to avoid exercise on the day of the test.

Excess Post-exercise Oxygen Consumption (EPOC)

The length of time that a subject's oxygen consumption remains elevated above baseline may vary with substrate utilization during exercise and recovery, adenosine nucleotide concentrations, catecholamines, thyroxine, cortisol, calcium ions, temperature (111), and possibly fitness level. A pre-exercise meal (of known caloric and macronutrient constitution) was scheduled at least four hours prior to exercise and was used to minimize possible confounding influences that may artificially elevate EPOC due to the thermic effect of food.

Hormonal parameters

Hormone measurements must be meticulously controlled so that the hormone levels are reflective of the response to be measured and do not involve the effects of extraneous influences. Cortisol exhibits the most dramatic variations due to circadian rhythm and, therefore, measurement conditions were controlled for the fluctuations in cortisol secretion. Cortisol displays a profound diurnal rhythm with highest concentration occurring in the early morning (approximately 6:00 am) and values in the evening being about half those in the morning (109). Response to stimulation may be smaller during the peak of circadian rhythm than during the trough (107). Therefore, cortisol is most accurately measured in the afternoon, after a four hour fast, with the subject rested for 30 minutes, and in abstinence of at least six hours of exercise (107,109,112). Resting cortisol can be overestimated if a fasted state and rested state are not controlled for; by residual effects of exercise on the day previous testing; by rise due to anticipation of exercise; and by not correcting for circadian variation when using the pre-exercise

method of measuring baseline (107,109). Cortisol response to exercise can be underestimated (and appear blunted) if resting levels are overestimated; exercise testing is scheduled in the morning; and if the exercise test duration is not sufficiently long to capture the delay in cortisol release (107,112).

Definitions

The operational definitions used in this investigation were as follows:

1. BMI = body mass index expressed as kg/m^2 . The BMI ranges used for classification were: normal weight 18 to 24.9 and obese 27.8 to 34.9.
2. Aerobic fitness = determined by $\text{VO}_{2\text{max}}$. This investigation included unfit males as defined by a score of $< 45 \text{ ml/kg/min}$. This categorization is based on normative data from Astrand (5).
3. EPOC = upon cessation of exercise, oxygen consumption remains elevated above pre-exercise testing levels. The end of EPOC was determined by a VO_2 that is equal to or below baseline values (33).
4. Ventilatory Threshold (VT) = The transition from predominately aerobic to anaerobic process during incremental exercise is known as the anaerobic threshold (AT), of which, VT is a parameter used to indicate the attainment of AT. Minute ventilation (VE) increases linearly with increasing workload, but when AT is reached, the rate of ventilation is greatly accelerated. Specifically, minute ventilation plotted against carbon dioxide exhaled (VCO_2) shows an uncoupled relationship at AT whereby VE is greater than VCO_2 at a given time or workload. This departure from linearity

occurs subsequent to the departure of $\dot{V}O_2$ from $\dot{V}E$ and is, therefore, the second threshold. The departure of $\dot{V}CO_2$ from linearity was used to denote $\dot{V}T$.

Purpose and Hypothesis

Purpose: To examine postexercise oxygen consumption (EPOC) across two groups of men distinguished by body fat.

Hypothesis: Compared to lean, obese subjects will have a diminished EPOC response to exercise.

Purpose: To examine metabolic hormone profile including cortisol, growth hormone, T_3 , and T_4 during recovery from exercise.

Hypothesis: During recovery from exercise, the induced rise in metabolic hormones will differ between groups with differing levels of body fat. The difference will be such that obese subjects are observed to have a blunted cortisol response, depressed growth hormone levels, and normal or depressed T_3 and T_4 .

Chapter 2: Literature Review

Introduction

Upon cessation of exercise, oxygen consumption ($\dot{V}O_2$) remains elevated above pre-exercise levels for a transient period of time. This phenomenon is called “excess post-exercise oxygen consumption”; also known as “EPOC”. Elevated oxygen consumption during recovery from exercise depicts heightened metabolic activity persisting beyond the removal of the exercise stimulus. The body continues to require energy to fuel the energetic costs of recovery, which involves replenishing substrate stores, restoring electrolyte balance, acid-base balance, temperature, and other processes disturbed by exercise (87). The underlying mechanisms responsible for EPOC are not well understood but explanations have focused on adenosine nucleotide concentrations, circulating catecholamines, thyroxine, cortisol, calcium ions, altered temperature, and substrate cycling (111).

EPOC has been suggested to play a significant role in weight control (9,10). Expending additional energy after exercise contributes to total energy expenditure, especially if EPOC is prolonged. The implication of a prolonged EPOC is that the prescription of exercise aimed at maximizing EPOC may not be the same combination of intensity and duration as prescribed to maximize caloric expenditure during exercise. However, many authors have concluded that the energetic cost of EPOC is very small and of little significance for weight control (20,33,41,48,62,67). As well, there may be a threshold for intensity and/or duration of exercise before a prolonged EPOC is achieved and this may be greater than the capability of the average person exercising for weight control. Nonetheless, recovery from exercise provides important information about how energetic challenges are differentially regulated and may lend insight into why some people are susceptible to weight gain and resistant to weight loss.

An efficient metabolism in the form of low energy expenditure has been described in the propensity toward obesity. Evidence of depressed resting metabolic rate (RMR) (8,89) and thermic effect of feeding (TEF) (58,116) in obese compared to non-obese population groups have been reported. If this is true, then EPOC, as a component of total energy expenditure, may also be blunted. Alternately, EPOC may be determined independently by exercise intensity and duration. If, however, EPOC is influenced by body fat level, it is not known if the duration or magnitude of EPOC (or both) is altered. In addition, during recovery from exercise, the induced rise in metabolic hormones may also vary in individuals with differing levels of body fat. Recent investigations into EPOC have attempted to further delineate factors that influence the magnitude and duration of EPOC, fast and slow components, and to identify the mechanisms and variables that influence EPOC.

I Background

EPOC is a term suggested by Gaesser and Brooks in 1984 (44) as an alternative to the earlier term “oxygen debt” introduced by Hill and Lupton in 1923 and refers to the “total amount of oxygen used after cessation of exercise in recovery therefrom” (44). The limitation of the term oxygen debt is that it suggests a cause-and-effect relationship. As explained in reviews by Bahr (10) and Gaesser and Brooks (44), the elevated oxygen uptake after exercise was thought necessary for the repayment of the oxygen deficit incurred during exercise. This was partitioned into a fast and slow component. The fast phase was attributed to oxidative removal of lactate in the muscle where it was formed, and the slow phase to oxidative removal of lactate, which had escaped by diffusion from the muscles (10,44). This arose from the observations of parallel appearance and disappearance of lactate and glycogen in a ratio related to the amount of oxygen consumed (44). In 1933 a revised theory by Margaria et al. (76) stated that only the

slow phase is linked to lactate metabolism. They demonstrated that decreased blood lactate did not occur until completion of the fast phase of recovery and, therefore, the rapid decrease in oxygen consumption must have been alactacid in origin. After Lundsgaard's (76) discovery of phosphagens and their role in muscle contraction, Margaria et al (76) hypothesized that the fast phase was attributed to the restoration of phosphagen levels as well as resaturation of myoglobin and hemoglobin with oxygen and the oxygen cost of ventilation and circulation (44,76). In 1984 Gaesser and Brooks challenged the oxygen debt hypothesis. They questioned the fate of lactate after exercise and the extent that lactate removal accounts for the increase in oxygen consumption. They contested that the oxygen debt is not necessarily equal to, or even correlated with, the magnitude of the preceding oxygen deficit or blood lactate and that a lack of a temporal relationship exists between decreasing blood lactate and the slow phase of the oxygen debt. The term EPOC was suggested as it does not imply causality between recovery and lactate. As well oxygen debt is associated with changes approximating an hour whereas EPOC allows for the inclusion of a more prolonged increase that can be observed for several hours after exercise (44).

II Factors influencing EPOC

Exercise Intensity

The factors influencing the magnitude and duration of EPOC include exercise intensity, exercise duration, and total energy expenditure (TEE). Exercise intensity and duration are considered the main determinants. When total energy expenditure (TEE) is held constant (thereby varying exercise duration), exercise intensities at 45, 55, and 65% $\dot{V}O_{2\max}$ (33); 65 to 80% $\dot{V}O_{2\max}$ (43); and 70 versus 105% $\dot{V}O_{2\max}$ (67) demonstrated a greater magnitude of EPOC at higher intensity compared to lower intensity exercise. Since the magnitude of EPOC followed consequent to intensity, it is determined,

therefore, more by the intensity of exercise rather than total work completed (48) or total energy expenditure (33). Intensity is also a greater determinant of EPOC than is exercise duration. When exercise durations of 20, 50, and 80 minutes were performed at 30, 50, and 70% $\dot{V}O_{2\max}$, no significant differences on EPOC magnitudes existed between the different durations until intensity equaled or exceeded 50% $\dot{V}O_{2\max}$ (48). The authors reported that the increase in EPOC magnitude as a result of increased duration was much smaller than the magnitude of increase as a result of increased intensity and that intensity accounted for a degree of variance on EPOC almost five times greater. Sedlock et al. (98) and Short and Sedlock (101) also found decreased recovery times in less intense compared to more intense exercise. The only study to oppose a positive linear relationship is by Chad and Quigley (24) who found larger EPOC magnitude at 50 versus 70% $\dot{V}O_{2\max}$.

Investigations delineating the relationship between exercise intensity and EPOC have used intensities ranging from 18% (20) to 108% $\dot{V}O_{2\max}$ (11). Generally, recovery oxygen consumption ($\dot{V}O_2$) is similar at low intensity exercise (20,99) or increases curvilinearly (20) until approximately 70% $\dot{V}O_{2\max}$, at which point EPOC increases disproportionately. Exercise below this threshold is associated with a single exponential decline in $\dot{V}O_2$ during the recovery period, whereas exercise at or above this threshold has a fast and slow component (51). Brehm (20) further contends that unless exercise intensity exceeds this threshold exercise has minor effects on EPOC magnitude unless it is of substantial duration, exceeding 60 minutes. In accordance, Sedlock (99) found similar EPOC magnitudes following exercise at 30 and 60% $\dot{V}O_{2\max}$.

That higher intensity exercise produces a greater magnitude of EPOC should not be surprising. In the high intensity condition, there will be a higher starting point in the recovery period and $\dot{V}O_2$ will have further to fall to return to baseline (33). As well, higher intensities present greater challenges to homeostatic regulation in temperature, acid-

base balance, and hormonal response (33). Although many studies have reported an intensity threshold phenomenon, these studies have not uniformly identified a particular intensity as being the threshold. As a speculated threshold level, 70% $\dot{V}O_{2\max}$ is the most referenced value found in the literature.

Exercise Duration

Investigations on the impact of exercise duration on resulting EPOC have been less concordant. Results are equivocal for whether or not there is an exercise duration mediated increase in EPOC. A positive relationship was not found when the exercise protocol consisted of four exercise sessions at 60 and 70% $\dot{V}O_2$ max for 20 and 40 minutes by Maresh et al. (75); and Freedman-Akabas et al. (41) did not document a sustained effect on $\dot{V}O_2$ when exercise duration increased from 20 to 40 minutes, performed at anaerobic threshold. In agreement with these results are protocols comparing continuous versus split-session exercise treatments (2,62). The sum of two 15 minute sessions produced a significantly greater magnitude of EPOC when compared to a continuous 30 minute session (2). As the authors propose, the 30 minute session was not sufficient to induce greater homeostatic disturbance beyond the 15 minute sessions. Possibly the majority of EPOC occurred during the first few minutes of recovery and EPOC during this time may not be affected by exercise duration (2,62). There may also exist an exercise duration threshold, supported by the fact that Quinn et al (87) found similar EPOCs for shorter exercise durations of 20 and 40 minutes. During longer periods, EPOC is more likely affected by exercise duration (2) but the idea of a threshold requires further investigation.

In contrast, a positive relationship was demonstrated by Bahr (10) and Chad and Wenger (25) when exercise intensity exceeded 50% $\dot{V}O_{2\max}$. These authors concluded a linear relationship between magnitude of EPOC and the duration of the exercise bout. In

Chad and Wenger's (25) protocol, EPOC increased 2.35 and 5.3 fold when exercise duration was increased from 30 to 45 minutes and from 30 to 60 minutes, respectively (both at 70%). In a similar protocol, Quinn (87) found a positive but non-linear relationship with an exercise bout at 60 minutes that yielded twice the EPOC as 20 or 40 minutes when intensity was set at 70%. Gore and Withers' (48) protocol consisted of a multiinteractive design crossing intensities of 30, 50, and 70% with durations of 20, 50, and 80 minutes and found that duration of exercise did not significantly impact EPOC until intensity equaled and exceeded 50%. The variability of these results may be due to the existence of a threshold of combination of exercise intensity and duration, below which the existence of EPOC is difficult to document (87).

III EPOC Magnitude and Duration

Although the existence of EPOC is well established, the details of its components, magnitude and duration, remains controversial. Magnitude of EPOC is the total accumulated oxygen consumption during the recovery period and duration of EPOC is the period of time that oxygen consumption remains elevated above baseline. Investigations into the factors influencing EPOC have primarily compared EPOC magnitudes. As discussed previously, there is a positive linear relationship between EPOC magnitude and exercise intensity, with perhaps an intensity threshold around 70% $\dot{V}O_{2max}$ at which point EPOC increases exponentially. There is less agreement over the influence of exercise duration on EPOC magnitude. A linear relationship may become apparent only when exercise intensity equals or exceeds 50% $\dot{V}O_{2max}$ or when exercise duration is prolonged past 60 minutes.

The effect of intensity and duration of exercise on EPOC duration is not clear. A wide range of EPOC duration is reported to exist, ranging from 3 minutes (42) to 48 hours (11,14,74). Whether a prolonged component actually exists is therefore

controversial. Many studies have demonstrated metabolic rate to return to pre-exercise conditions within the first hour after exercise while other studies have demonstrated EPOC durations beyond one hour.

Studies that do not support a prolonged EPOC response have reported a return of $\dot{V}O_2$ to resting levels within 60 minutes after the end of exercise, regardless of the intensity or duration of the exercise bout. In a study by Dawson et al. (37) using graded intensities EPOC duration was similar and very short (<15 minutes) despite an EPOC magnitude four to five times higher after high intensity (65%) compared to low intensity (45%, 55%) exercise. The authors concluded that with exercise energy expenditure held constant, the intensity of exercise affects the magnitude but not the duration of EPOC. Sedlock (99) also failed to produce a prolonged EPOC after 850 kJ work at 40 and 60% $\dot{V}O_{2max}$ and concluded that the duration of EPOC seemed independent of the chosen intensities. At exercise intensities greater than the hypothesized threshold the various combinations of intensity and duration in two studies also did not produce a prolonged EPOC (75,98).

When a prolonged EPOC is demonstrated the exercise protocols have consistently involved intensities equal to or exceeding 70% $\dot{V}O_{2max}$. The exceptions that exist occurred when exercise duration is very prolonged such as exercise at 50% $\dot{V}O_{2max}$ for 80 minutes (48) or 50% $\dot{V}O_{2max}$ for 3 hours (14). Among the investigations using similar protocols at approximately 70% $\dot{V}O_{2max}$, disparate results have been demonstrated. Two comparable protocols using an intensity of 70% for 45 and 60 minutes (25) and for 50 minutes (48) generated EPOC durations of 204, 455, and 240 minutes, respectively. On the other hand, similar exercise protocols have also generated different EPOC durations. At an exercise intensity of 70% for 80 minutes, EPOC duration is reported to last 480 minutes shown in one study (48) and to exceed 12 hours shown in two other studies (10,74).

A discrepancy also exists between the combinations of magnitude and duration of EPOC. As reported by Laforgia et al. (67): a magnitude of 14.6 and 15 liters of oxygen were documented over 8 and 9 hours, respectively, while a magnitude of 16.3 liters was sustained for 14 hours above baseline. Therefore, similar magnitudes of EPOC were associated with dissimilar durations. Furthermore, in one study, under the same exercise treatment, two subjects required up to 80 minutes to recover while the rest of the subject pool recovered under 60 minutes. The authors concluded that some type of interaction between exercise intensity, duration, and training status occurred to prolong metabolic recovery in selected cases. However, no clear pattern has emerged relating this interaction (101).

IV Fast & Slow Components

Various durations of EPOC reported for an equivalent magnitude (liters) of EPOC or exercise treatment can be explained, in part, by the kinetics of $\dot{V}O_2$ decline. As mentioned previously, recovery $\dot{V}O_2$ comprises a fast and slow component. Difference in the kinetics of the fast phase is demonstrated after graded exercise intensities (33). EPOC magnitude was greater after the high intensity compared to moderate or low intensities but EPOC duration remained similar and very short (<15 minutes). The rate of decline in oxygen consumption towards baseline was faster for the high intensity condition, which led to a similar EPOC duration as the other exercise conditions. This difference mainly arose from the fast component, with little difference in the slow phase of recovery (33).

This pattern of $\dot{V}O_2$ kinetics also emerges when comparing trained versus untrained subjects (43,51,101). Training results in smaller $\dot{V}O_2$ accumulation in response to exercise at the same absolute workload (43,51). This is in part attributed to the fact that a given absolute workload represents a smaller fraction of $\dot{V}O_{2max}$ after training (43).

There is both a smaller absolute $\dot{V}O_2$ at the start of recovery and a faster rate of recovery (51). Furthermore, Hagberg et al. (51) concluded that training resulted in almost complete elimination of the slow component of recovery.

A faster rate of recovery from the same relative workload also results from training since a higher threshold is required to elicit the lactate threshold and key hormonal responses. EPOC magnitude might be expected to be larger for trained subjects following exercise at 70% $\dot{V}O_{2max}$ because of a higher $\dot{V}O_2$ starting point. But Short and Sedlock (101) reported no difference between groups. The explanation stems from the faster initial rate of decline for trained subjects, which quickly led to similar $\dot{V}O_2$ values in each group. Similarly, Frey et al. (43) reported equivalent magnitudes of total EPOC between subject groups after low intensity and high intensity exercise set at 300 kcal. Although trained subjects demonstrated a higher $\dot{V}O_2$ at the start of recovery, a greater fast EPOC component following both intensities led to a shorter EPOC duration. However, not all such studies have been able to demonstrate such a training effect on EPOC. Sedlock (100) found that metabolic adaptations resulting from endurance training are not sufficient to impact EPOC. The reason for these discrepancies remain unknown, but exercise intensity and duration used may be partly responsible.

V Mechanisms

The explanation for EPOC may be found at the level of the mitochondria since this organelle is the site of oxygen consumption in the cell (44). Direct control of mitochondrial respiration may be altered by concentrations of adenosine triphosphate, adenosine diphosphate, phosphate, and creatine phosphate (44). In a review by Gaesser and Brooks (44), the kinetics of EPOC and creatine phosphate were reportedly similar. The time-course of creatine phosphate resynthesis after exercise, like EPOC, was biphasic, exhibiting a fast and slow component (44).

Although post exercise oxygen consumption will likely be controlled directly by intramitochondrial phosphate levels, kinetics of the EPOC phenomenon are not confined to this compound, as a variety of additional factors have been shown to exert an influence on mitochondrial respiration (44). Indirect mechanisms such as the replenishment of oxygen stores in blood and muscle; lactate removal; and increased temperature, circulation, and ventilation, known as the Q10 effect (18), are thought to contribute to the rapid component of recovery. These processes are referred to as rapid since they are thought to be completed within the first hour of recovery. The slow phase is attributed to the indirect mechanisms that may affect mitochondrial respiration such as substrate cycling, calcium ion concentrations, and hormones (catecholamines, thyroxine, and cortisol) (18). Alternately, both the fast and slow phases of EPOC may be governed by exactly the same mechanisms, but as the relative contribution of each factor changes so does the rate of metabolism (44).

Temperature

Increased body temperature during exercise has been suggested as a factor partially responsible for EPOC by increasing cellular respiration and decreasing phosphorylative coupling efficiency in the mitochondria (44). In agreement with the Q10 hypothesis, Gore and Withers (48) found that rectal temperature remained significantly elevated above resting level for one hour following 20 minutes of exercise performed at 70% $\dot{V}O_{2\max}$ and for 5 hours following 50 minutes of exercise at a similar intensity. Frey et al. (43) compared exercise bouts of graded intensities and reported that subjects took longer to return to normal resting temperature following high intensity compared to low intensity exercise. Gaesser and Brooks (44) attribute temperature as the most important factor affecting EPOC and Hagberg et al. (51) calculated temperature to account for 60 to 70% of the slow component.

There is also evidence discounting the Q10 hypothesis. Quinn et al. (87) found no significant difference in body core temperature between three exercise bouts of varying durations. In a protocol by Laforgia et al. (67), which compared two bouts of exercise at submaximal and supramaximal intensities, rectal temperature returned to control levels within two hours in both conditions despite a significantly greater EPOC associated with the supramaximal treatment. The authors reported the correlation and variance between temperature and EPOC for the submaximal treatment to be 0.33 and 9% and for the supramaximal treatment to be 0.13 and 2%, respectively (67). Frey et al. (43) reported that temperature is highly correlated with EPOC ($r=.98$) but that it accounted for only 16 to 24% of the slow component across subject groups and exercise intensities. Furthermore, in this study, rectal temperature had returned to resting levels within 40 minutes of recovery following low intensity exercise in trained subjects despite the observation that EPOC remained elevated. Therefore, factors other than core temperature must be responsible for EPOC beyond 40 minutes.

It has been suggested that muscle temperature may be more closely correlated with EPOC. This was not measured in any of the studies mentioned. However, when measured in rats, muscle temperature affected mitochondrial energetics by increasing nonconservative (state 4) respiration and decreasing phosphorylative coupling efficiency. As a consequence, more oxygen is consumed for a given amount of ATP to be synthesized (44). As well temperature elevation would affect other processes such as increasing substrate cycling rates (20).

Lactate Removal

Originally it was thought that the removal and conversion of lactate accounted for the observed EPOC. It is clear now that it only partially accounts for the total amount of EPOC. Challenge to the oxygen debt theory contests its underlying assumption that a

major part of the lactate formed (approximately 80%) during exercise is converted to glycogen and the rest is oxidized to supply ATP for this purpose. The primary fate of lactate formed after exercise is now thought to be oxidation (43,44) while only about 20% is converted to glycogen. As well, the original hypothesis is thought to be too simplistic. A number of studies have shown the magnitude of the oxygen debt is not necessarily equal to or even correlated with the magnitude of the preceding oxygen deficit or blood lactate concentration (44). Furthermore the time-course of lactate removal and EPOC have been shown to be discrepant. The temporal relationship between EPOC and lactate levels has presented contradictory evidence whereby lactate recovery precedes $\dot{V}O_2$ recovery as well as $\dot{V}O_2$ recovery preceding lactate recovery. In a review by Gaesser and Brooks (44) on the metabolic basis of EPOC, the authors refer to earlier studies using prolonged exercise, which led to elevated EPOC without any concomitant increase in blood lactate. As well, experiments at altitude have demonstrated higher lactate levels post exercise combined with lower EPOC compared with sea-level (44). Dietary modifications also demonstrate similar EPOCs associated with different lactate levels, for instance during the glycogen depleted state (44).

Contemporary views on lactate metabolism put forth by Gaesser and Brooks (44) contend that lactate production can occur even when the muscle is well oxygenated and can be influenced by factors other than the degree of tissue oxygenation. Lactate metabolism is occurring at all times and thus cannot be associated with a particular phase of EPOC. At rest, lactate oxidation may account for as much as 15% of resting metabolic rate (44).

Substrate cycling

Increased fat oxidation and concomitant increase in triglycerol-fatty acid (TG-FA) cycling are hypothesized to make significant contributions to EPOC (80). Recovery from

exercise is associated with a shift from carbohydrate to fat as a fuel substrate and since the oxygen cost of fat catabolism is greater than carbohydrate catabolism, more oxygen is consumed (80). Also in support of this hypothesis, caffeine ingestion prior to exercise, which is stimulatory to fatty acid mobilization and oxidation, and nicotinic acid ingestion which is inhibitory to fatty acid metabolism, significantly increased and decreased, respectively, the magnitude of EPOC when measured for one hour following exercise (111). Increased fat oxidation perpetuates increased rate of energy requiring TG-FA cycling after prolonged exhausting exercise. Increased rate of cycling has been found after low intensity, long duration exercise such as after 2 hours at 50% $\dot{V}O_{2\max}$ (9) and after 4 hours at 40% $\dot{V}O_{2\max}$ (121). Isotopic tracers have demonstrated that approximately 50% of EPOC can be accounted for by an increased rate of TG-FA cycling (9). Newsholme (80) suggests that substrate cycling would have to operate at only 3-17% of maximum capacity to account for a $\dot{V}O_2$ of approximately 1 ml.min⁻¹ kg⁻¹. Rates of cycling are increased before and after exercise, during absorption of a meal, during stress, may depend on previous energy intake and expenditure, and may be under hormonal and nervous control (80). Furthermore, there may be an interactive effect with other mechanisms. Fatty acids may also influence the coupling of oxidative phosphorylation in the mitochondria in white adipose tissue (74) and may be the cause of elevated temperature observed for some hours after exercise (80).

The primary role of such cycles is probably in metabolic regulation as they provide one mechanism for increasing the sensitivity of the regulatory mechanism of metabolism (80). A substrate cycle involves two opposing nonequilibrium reactions that are catalyzed by different enzymes and that operate simultaneously. At least one of the reactions hydrolyze ATP and there is no net conversion of substrate to product (9). This is also referred to as futile cycling since these cycles involve an obligatory loss of chemical energy as heat. The greater the rate of cycling, the greater the rate of

conversion of chemical energy to heat. Control of this rate is the aspect of cycling that permits maintenance of normal body weight by dissipating excess energy as more oxygen is consumed for a given amount of ATP produced. An increased sensitivity is achieved because, at rest, the net flux through one reaction is decreased to almost zero as a result of its opposing reaction. A small increase in the concentration of regulators above resting levels produces a very large increased net flux through the direction of its reaction (like a threshold response). The higher the rate of cycling as compared with the net flux in the resting state, the better the threshold response, and hence the greater the improvement in sensitivity. That is, sensitivity is proportional to the ratio of cycling rate to flux (80).

Respiratory exchange ratio (RER)

The post exercise shift toward fat metabolism is associated with a RER lower during recovery compared to rest (24,111). Bahr (10) has hypothesized that exercise may in some way make fat tissue more receptive to stimulating increases in fat metabolism and since trained subjects are known to have a higher oxidative capacity, then greater fat utilization, lower RER, and greater magnitude of EPOC following exercise would be expected. Not all such studies have been able to corroborate a lower RER during recovery. Investigations by Brehm (20), Freedman-Akabas et al. (41), Sedlock (100), Frey et al. (43) and Short & Sedlock (101) all report no difference in RER between fit and unfit subjects when investigated under a variety of exercise protocols. RER is often higher for untrained subjects during exercise, but the same or lower than trained subjects during the recovery period (24,43,111). This is consistent with the observation that EPOC is greater in untrained than trained subjects, but inconsistent with the idea that training results in a greater reliance on fat during recovery. One possible explanation for this result is that at the same relative $\dot{V}O_{2\max}$, muscle glycogen stores

would be depleted to a greater extent in untrained compared to trained subjects, thereby leading to a greater reliance on fat metabolism by untrained subjects during the recovery period to replenish these stores.

Hormones

Since hormones regulate fuel metabolism, changes in RER likely accompany changes in the metabolic hormones. Catecholamines, thyroxine, cortisol, and growth hormone (GH) are stimulatory to lipolysis and would thus increase EPOC. These processes are stimulated during exercise and only slowly decline to resting levels after the cessation of exercise, even if there is no further increase in secretion during the recovery period (18). Therefore, there is a hormone-induced effect that may last beyond the exercise period. During exercise, the magnitude of increase in cortisol, GH, thyroid hormones, and catecholamines is related to exercise intensity and duration. Relating the magnitude of EPOC with elevated hormone levels during recovery, although based on a limited number of studies, have focused on the influence of hormones upon mitochondrial respiration.

Mitochondrial Respiration

Hormones probably act on mitochondrial respiration indirectly by stimulating ATP-requiring processes in the cell. Norepinephrine (NE), thyroxine and cortisol are thought to enhance sodium-potassium ($\text{Na}^+\text{-K}^+$) pump activity (44,87) by increasing cell membrane permeability to Na^+ and K^+ , which would subsequently lead to increased ATP production and oxygen consumption (43,44). The uncoupling of oxidative phosphorylation consequently leads to increased state 4 mitochondrial respiration whereby more oxygen is consumed for a given amount of ATP that is produced. This

effect on the electron transport chain could strongly influence both the fast and slow components of EPOC (43).

Mobilize fuel reserves

The enzymatic activities of metabolic fuel provision are orchestrated by hormones. Catecholamines, cortisol, GH, and thyroxine mediate the regulation of fat and carbohydrate mobilization and oxidation and are associated with increases in energy expenditure.

Catecholamines

Catecholamines stimulate glycolysis, gluconeogenesis, and lipolysis (18) and they are a marker of sympathetic activity and energy expenditure (43). Infusion of relatively low doses of adrenaline stimulates energy metabolism in humans (18) and may be an important regulator of triglycerol-fatty acid (TG-FA) cycling. Catecholamines increase free fatty acid (FFA) concentration in plasma, which is stimulatory for increased FFA reincorporation into adipose tissue and uptake into muscles; thus perpetuating the TG-FA cycle.

During exercise the concentration of plasma catecholamines increase linearly with exercise duration and exponentially with exercise intensity, which is the same relation as that between EPOC and exercise intensity and duration (18). Frey et al. (43) found a moderate correlation ($r=.78$) between NE and EPOC, especially during the rapid phase and initial phase of the slow components. The return of oxygen consumption and plasma catecholamines to resting levels, however, does not follow the same time course. Borsheim et al. (18) reviewed earlier published data and concluded that in all cases where catecholamines and EPOC have been measured, the increased oxygen uptake outlasted the increase observed in catecholamine concentration. However, it

remains possible that the sensitivity of adipose tissue to catecholamines may increase after exercise. A 20-35% increase in adipocyte lipolytic responsiveness to catecholamines after exercise has been reported (18). Thus, it appears likely that the observed increase in TG-FA cycling is at least partly due to release of and responsiveness to catecholamines post-exercise and that plasma catecholamine concentration may be an insensitive indicator of sympathetic activity (18). But Borsheim et al. (18) concede that some pilot studies in humans have not shown any difference in EPOC after β -antagonist administration or increased sensitivity for catecholamines for many hours post-exercise.

Cortisol

The primary function of cortisol is to mobilize fuels and maintain adequate blood glucose levels. It does this by inhibiting glucose uptake into muscle and fat cells, thereby causing them to catabolyze their own fuel sources (amino acids and free fatty acids), which are released into the bloodstream and then sequestered into the liver and used as substrates for gluconeogenesis. Secretion of cortisol occurs in response to food intake, stress, and exercise (106). In response to exercise, cortisol progressively increases according to intensity and duration and is influenced by an individual's fitness level. There may be an exercise threshold of intensity ($> 60\text{-}70\% \text{ } V_{O_{2\max}}$) and duration (>20 minutes) whereby cortisol excretion increases significantly in the post exercise period and this may relate to the non-linear changes observed in EPOC (35,111).

Growth Hormone

The function of GH in terms of energy metabolism is to stimulate lipolysis and gluconeogenesis, and inhibit glucose uptake by muscle and adipose tissue. Stimuli for GH secretion include exercise, stress, fasting, low plasma glucose levels, and sleep.

The GH secretion that occurs during sleep accounts for the majority of the total 24 hour secretion of GH. During the day there is little or no GH secreted, although bursts may be elicited by the above mentioned stimuli (115). During exercise, levels of GH increase in response to intensity and duration, with more pronounced increases occurring with greater intensities and durations of exercise (40). The increase, however, is not immediate but occurs gradually with time and is too slow to account for the rapid stimulation of lipolysis at the onset of exercise (88). As well GH levels off at exhaustion, which is when FFAs are most in demand (88). The activity of GH during recovery, however, has not been elucidated, but may relate to the anabolic properties of the hormone.

Thyroid Hormones

The thyroid axis involving thyroxine (T4), triiodothyronine (T3), and reverse triiodothyronine (rT3) is affected by energy intake and output and, thus, provides a marker of energy status. However, the axis is buffered against sudden changes and the level of these hormones in response to acute exercise is, therefore, unlikely to change (32,40). Nonetheless, thyroid hormones alter aerobic metabolism by influencing lipolysis by potentiating adrenoreceptor sensitivity to catecholamines (4) and by altering mitochondrial respiration. Skeletal muscle is a primary target organ for thyroid hormones (52) and T3 receptors have been found on the mitochondrial membrane. This implies that thyroid hormones may regulate oxidative metabolism (53). As well, these hormones are suggested to affect the expression of genes that encode for mitochondrial enzymes, notably cytochrome C oxidase and succinate dehydrogenase (53).

When thyroid hormone levels are below clinical significance, lower circulating levels cannot be directly attributed to lower muscle aerobic capacity (52). However, comparisons between subjects with varying thyroid hormone status have found that

lower thyroid levels are associated with impaired oxidative capacity (53). At a given level of work, increased anaerobic contribution during exercise (indicated by an elevated ratio of inorganic phosphate to phosphocreatine) and slower recovery kinetics of these compounds to baseline post exercise (53) would indicate less control of aerobic metabolism and mitochondrial respiration during exercise (52) and extended recovery (53) post exercise.

VI Variables influencing EPOC

Different methodological protocols used by different investigators have made findings on EPOC difficult to compare. In addition to differences in exercise intensity and duration, other factors that might help explain discrepant results include: fitness level, intensity of the exercise treatment relative to the subjects anaerobic threshold, interaction with dietary induced thermogenesis (TEF), and body composition (75). As well, criteria for establishing the end of EPOC, measurement of resting metabolic rate (RMR), and recovery posture used are not standardized across studies.

Training Status

Aerobic training is an important consideration when examining EPOC since physiological adaptations of trained individuals alter the regulation of energy metabolism. Cardiovascular and muscular adaptations with training improve the efficiency of metabolic regulation: temperature regulation is improved, lactate clearance is enhanced, hormonal response is decreased, muscle blood flow is increased, and substrate utilization is enhanced, leading to better control of metabolic stress during and after exercise (43). This has led to the hypothesis that after training, the $\dot{V}O_2$ kinetics at both the onset and offset of exercise is accelerated resulting in a smaller oxygen deficit and debt, respectively (51). This is in part attributed to a training-induced increase in

$\dot{V}O_{2\max}$ so that a given absolute workload represents a lower percentage of $\dot{V}O_{2\max}$ in the trained individual. Thus, the lower relative stress imposed by exercise elicits a reduced or attenuated response in many of these variables. Training also increases the relative workload required to elicit the lactate threshold and key hormonal responses such as the elevation in catecholamine levels (43,51). Adjustment to the energy requirements of work and the rate of recovery thus occurs more rapidly at a given relative intensity.

To date a consensus has not been reached regarding the effect of physical training on EPOC magnitude or duration. Inconsistent findings are reported in literature. Findings by Short and Sedlock (101), Frey et al. (43), and Hagberg et al (51) support the hypothesis that exercise training increases a subject's rate of recovery from exercise. Following exercise bouts of equal relative or absolute workrate, trained subjects had shorter EPOC durations than untrained subjects despite greater or equivalent EPOC magnitudes (43,51). This indicates faster regulation of post-exercise metabolism. Data from longitudinal studies also demonstrate faster metabolic recovery from exercise after untrained subjects were exposed to a 9 week training program (51). However, not all such studies have been able to demonstrate such a training effect on EPOC. Investigations by Brehm (20), Freedman-Akabas (41), and Sedlock (100) all report no difference between fit and unfit subjects when investigated under a variety of exercise protocols. Sedlock (100) concluded that EPOC is not compromised by differences in body weight or fitness level and Freedman-Akabas et al. (41) documented this finding across fitness categories. Furthermore, Chad and Quigley (24) showed contrary findings and suggested that aerobic training is associated with larger recovery energy expenditure and potentially longer EPOC duration at a given relative intensity. Although the majority of findings do not support this trend, this position is somewhat justifiable. Since trained subjects are known to have a higher oxidative capacity, then greater fat utilization, lower RER, and greater magnitude of EPOC following exercise can be

expected. The caffeine and nicotinic acid trials mentioned above would also suggest this trend (111). Nonetheless, there is greater support for the finding that training is associated with faster post-exercise energetics.

Anaerobic Threshold

Since the anaerobic threshold (AT) for most people falls at approximately 60-70% $\dot{V}O_{2\max}$ (101), and it has been postulated that there exists a threshold at about this intensity, above which elicits a prolonged EPOC, the question arises of whether or not a prolonged duration of EPOC is triggered by activity above the AT. At higher intensity exercise, factors such as lactate metabolism, increased tissue temperature, lower high-energy phosphate concentrations, and greater reliance on carbohydrates differentially challenges the regulation of temperature, substrate utilization, and hormone release (101). When subjects were exercised at intensities below AT (33) very brief durations of EPOC were documented and when the exercise treatment was set at or above 70% $\dot{V}O_{2\max}$, possibly above AT of the untrained subjects (43,101), EPOC durations of the untrained subjects exceeded the trained counterparts. If the explanation is correct, that the 70% workload imposed a greater stress on untrained than trained subjects because of differences in lactate threshold, then adjustment of the workload relative to each individual's lactate threshold would likely increase the homogeneity of the EPOC response (43,101).

Recovery Posture

Alternatively, an explanation for what elicits a prolonged EPOC may be an artifact of experimental design rather than reflective of the exercise stimulus under investigation. There is disagreement over the effect on EPOC of adopting a recumbent versus upright sitting posture during recovery. Dawson et al. (33) suggests that blood

flow to and from the active muscles is facilitated by adopting a prone or supine position and stroke volume is maximized, thereby possibly shortening the slow phase of recovery and possibly accounting for the speedy return of $\dot{V}O_2$ to baseline. On the other hand, Short et al. (101) suggest that longer EPOC occurs in the supine versus upright sitting posture as a result of the slight reduction in resting metabolic rate (RMR).

RMR

Inadequate control for factors known to influence RMR can confound comparisons between studies. RMR represents the energetic cost of maintaining the integrated functions of the body at rest. This includes the regulation of temperature, cardiovascular system, pulmonary system, electrolyte gradients, central nervous system, and chemical reactions. RMR accounts for 60-70% of total energy expenditure and is affected by age, gender, body size and composition, body temperature, thermogenic hormones, physical activity, genetics, and food intake. Intraindividual biological variability in RMR is approximately 5% (67). RMR is known to vary diurnally and, thus, temporally matched measurements of each subject's RMR on the control day and before the exercise treatment is necessary (48,67). Not all EPOC studies have controlled for diurnal variation or did not document it.

Criteria for establishing the end of EPOC

Recovery oxygen consumption is completed when oxygen consumption equals preexercise levels (baseline) but the criteria for establishing the end of EPOC are inconsistent among investigators. Within the literature, baseline recovery is often taken as two or more consecutive minute values at or below the baseline resting $\dot{V}O_2$ or within one standard deviation of the resting $\dot{V}O_2$ (33). This latter baseline is less stringent and would result in a shortened recovery time that underestimates the EPOC (33).

Thermic Effect of Feeding (TEF)

One factor that should be considered in the studies that show a prolonged EPOC is the potential interaction with diet-induced thermogenesis. This thermic effect of food (TEF) is caused by the energy requirements of digestion, absorption, transportation, and storage (48). A high TEF expends ingested calories for its own digestion and results in fewer calories being left over for storage. Conversely, a low TEF results in more calories being available for storage (58). TEF is affected by diet composition (carbohydrates have a high TEF whereas fats have a low TEF) and is potentiated by exercise (58). A reduction in postprandial thermogenesis has been described in obesity (58,116), although contradictory reports of normal TEF also exist.

Three of the previously mentioned studies that report a prolonged EPOC (12,14,74) had meals included in the post exercise period. Although they attempted to control this by having a no-exercise, meal-only trial, the interaction between meal and exercise on post exercise metabolism could not be analyzed. It is only possible to precisely measure the TEF against baselines with exercise-no food and rest-no food (48). Discrepant findings of EPOC magnitude and duration may be, therefore, partly attributed to dietary induced thermogenesis.

Body composition

The influence of body composition on EPOC has not been investigated. An efficient metabolism in the form of low energy expenditure has been described in the propensity toward obesity. Evidence of depressed RMR (8,89) and TEF (58,116) in obese compared to non-obese population groups has been reported. Therefore, if obesity is associated with diminished RMR and TEF, it seems reasonable to predict a reduced EPOC.

The mechanisms of a blunted oxidative potential in EPOC may focus on lactate, RER, and the hormones cortisol and GH. Increased glucose oxidation relative to fat oxidation is hypothesized in obesity (6,29,39,71,83,114). Gluconeogenesis is partially fuelled by lactate generated during exercise and obesity may be associated with a greater flux through this cycle, resulting in lower lactate concentrations measured during the recovery period (104). Alternately, a blunted oxidative capacity may lead to lower lactate clearance and higher plasma levels measured post-exercise. However, the literature does not agree on whether lactate levels are different in obesity. Investigations using oral glucose ingestion, overnight fasting, and exercise have found blood lactate levels that are higher (93), lower (68,104), and similar (3,103,116) between obese and lean subjects. Neither do RER values help to clear up these discordant results. A number of studies have reported no significant correlations between body fatness and substrate oxidation when evaluated by RER (36,46,54), which is in contrast to the long held notion linking body fatness and muscle fibre-type profile to substrate oxidation (117).

A greater tendency toward glucose metabolism is in contrast to the idea that increased levels of circulating free fatty acids, particularly evident in visceral obesity, result in increased fat oxidation and decreased glycogen breakdown. Although short-term increases in lipid availability through feeding or infusion of long and medium chain fatty acids (31,78) are associated with greater fat oxidation, chronic exposure to high circulating levels of fatty acids is not associated with increased fat oxidation (66,82,113). In obesity, fatty acid oxidation during post absorptive conditions is diminished within skeletal muscle while esterification and storage of lipids is increased (64). Excessive levels of plasma lipids tend to flood the portal circulation at metabolically inappropriate times when fatty acids are unlikely to be oxidized, thus exposing tissues to excessive free fatty acid levels, promoting lipid accumulation and giving rise to insulin resistance

(78). As well, some but not all studies have found impaired ability in obesity to use fat as a fuel during exercise (61,89). But there is further disagreement as to the source of lipid oxidation, with some studies suggesting reduced use of plasma non-esterified fatty acids (61,95,96) and other studies reporting normal oxidation of plasma sources but reduced non-plasma utilization. Furthermore, during recovery from exercise, there may be an increased re-esterification of FFA related to greater lipoprotein lipase (LPL) expression in adipose tissue of the obese (59,91).

Among the endocrine changes in obesity, a diminished response of GH to stimuli and resting hypercortisolism (65) may both impact upon mitochondrial energetics and lower the EPOC response. It is well known that GH secretion is impaired in obese individuals. A central point of debate about the insufficiency of GH in obesity is whether it represents an adaptive phenomenon (i.e. in response to the accumulation of visceral fat) or if it reflects a true impairment of the axis activity (72). Although GH activity is mainly under neuro-endocrine control, metabolic influences such as by FFA and leptin can greatly modulate its function. The pathogenesis of GH insufficiency, as reported by Maccario et al. (72), could originate centrally (CNS-related) and hypothetically include increased somatostatin release or impairment of GHRH tone or peripherally and involve metabolic alterations in insulin, FFA, leptin, and/or glucose.

Concerning the role of metabolic alterations, modulation of GH activity is achieved through pharmacological interventions with heparin and acipimox and through diet-induced changes in adiposity. Normalization of GH secretion to GHRH is shown with administration of acipimox, a lipolysis inhibitor, leading to decreased levels of circulating FFA and with diet-induced weight loss. Diminished response of GH to GHRH in obese humans is associated with infusion of heparin causing increased levels of FFA and also associated with overfeeding in rats. The determination of GH secretion by manipulation of FFA levels suggests the hypothesis that somatotroph deficit in obesity is

functional and reversible by weight loss (although full restoration of somatotroph secretion comparable to the response achieved in lean controls has not been shown). Chronic elevation of free-fatty acids is known to have an inhibitory effect on somatotroph secretion (28,72,73), possibly over cell membrane depolarization at the pituitary level (72).

Although cortisol shares similar and redundant functions to GH (both stimulate gluconeogenesis and are lipolytic), the extremes of these hormone states, as seen in disease, show that it is with a deficiency of GH but an excess of cortisol (Cushing's Syndrome) that is associated with obesity. The explanation at the adipocyte level may be that GH mobilizes FFAs through the hormone-sensitive lipase, while cortisol, although lipolytic, also exerts the opposite effect of promoting lipid accumulation by expressing LPL activity (15). Thus, it seems likely that GH promotes FFA's as a preferential fuel source, whereas cortisol favours glucose as a primary substrate. The low levels of GH found in obesity is then coupled with decreased reliance on FFA for energy metabolism and increased reliance on glucose. Accordingly, during GH infusion, there is a suppression of endogenous (hepatic) glucose production and decreased glucose infusion rate (M value), which is likely secondary to the increased lipid availability and subsequent substrate competition (Randle Cycle). As well, the deprivation of GH therapy in GH deficiency induced a nocturnal increase in levels of gluconeogenic substrate precursors (60).

VII Implications

Responsiveness of the metabolic system to changing energy demands is differentially regulated in healthy weight compared to obese population groups. Comparisons between groups reveal varying biochemical and hormonal profiles at rest, in response to exercise, and during recovery from exercise. Differences in the

mechanisms controlling energy balance between obese and non-obese populations are such that a permissive metabolic environment may be causal to obesity. Increased metabolic efficiency in the form of low energy expenditure, a tissue enzymatic profile favoring lipid storage, altered neurohormonal control over metabolism (4,11), a reduction in nonconservative mitochondrial respiration, and decreased rate of TG-FA cycling (39) have been described in the propensity toward obesity. Control of these mechanisms of metabolic regulation is hypothesized to permit maintenance of normal body weight by dissipating excess energy in the form of heat. If this is correct, then obese subjects may be observed to have a diminished EPOC response to exercise. Furthermore, it may be during recovery, rather than during exercise, that differences in how the body copes with changing energy demands and prioritizes metabolic fuel selection are most apparent. Therefore, the purpose of this investigation was to examine postexercise oxygen consumption (EPOC) and metabolic hormone profile during recovery from exercise in a comparison between groups of people distinguished by amount of body fat.

Chapter 3: Methods and Procedures

Overview of study

When subjects arrived in the lab, they were given an orientation of the testing areas as well as an overview of the research study (Figure 1). A consent form was administered to each subject. A date was then set for a second visit at which time body composition was determined by hydrostatic weighing and the $\dot{V}O_{2\max}$ test was administered. A diet record was handed out and instruction on proper record keeping given in verbal and written form. The next visits were for the Baseline and Exercise/EPOC sessions (Figure 2). The Baseline session was scheduled in the afternoon and $\dot{V}O_2$ and blood samples were collected to establish resting values. The Exercise/EPOC session was scheduled at an identical time to Baseline and within 1 week of the first measurement (or as soon as permitted). The circadian baseline method as described by Thuma et al. (107) recommends measuring a hormonal profile on a day separate from the exercise intervention but under the same conditions to establish a reference baseline. Typically, investigators will assess pre-exercise values and use these as a point of comparison for an exercise response. This protocol neglects the circadian rhythm of cortisol and may underestimate the cortisol response to exercise.

Research design

This study was conducted as a cohort analytical study. Comparison of different cohorts was made to examine the postexercise oxygen consumption and metabolic hormone profile across groups distinguished by body composition. Two cohorts classified according to percentage body fat were examined to answer the questions: is

obesity (independent variable) associated with diminished postexercise oxygen consumption (dependent variable)? As well, is the metabolic hormone response during recovery from exercise (dependent variable) different between the obese and lean groups (independent variable)? A hormonal profile was examined since hormones are known to exert influence over the enzymatic activities of metabolism and overall energy balance. A favorable hormonal response to exercise that is conducive to fat oxidation includes an increase in growth hormone and cortisol with intensity and duration of exercise and slight increase or no change in thyroid hormone levels.

Subjects

The sample of participants examined was men aged 30 to 39 years. Volunteers from the city of Edmonton were recruited using advertisement with posters at various locations throughout the city and local media. Initial screening for entry into the study included BMI (kg/m^2) in the range of 18.5 to 24.9 to represent a normal weight group and 27.8 to 34.9 to represent an overweight group. This classification is based on current interpretation of BMI described in health and medical literature as discussed in Chapter 1. BMI was used only in initial recruitment to quickly screen and exclude subjects who were clearly outside the range of the two groups. The criterion for entry was based on percentage body fat. For the purposes of this study subjects were excluded if they fulfilled the following:

- reported history of diabetes, hypertension, heart disease, and other metabolic disturbances
- medications known to influence metabolism or adipose tissue mass
- smoking
- weight instability within last 3 months described by a change of at least 3 kg
- BMI >34.9 since this level of adiposity is more likely associated with metabolic disorders (19)
- Inability to exercise (orthopedic limitations, etc.)
- aerobically trained as defined by $\text{VO}_{2\text{max}} \geq 45 \text{ ml kg}^{-1} \text{ min}^{-1}$

These conditions are known to influence metabolism and would consequently confound the analysis of hormonal data. Subjects included only males since gender differences also confound hormonal profile. Subjects were assessed for the following measures: BMI, aerobic fitness (maximal or peak oxygen consumption), 3 day diet record, body fat by hydrostatic weighing, EPOC (post-exercise oxygen consumption), and hormonal parameters including cortisol, growth hormone, and thyroid hormones (T_3 , T_4). Subjects were classified into groups based on percentage body fat (independent variables) and assessed for differences in EPOC response (dependent variable).

Subjects were classified by percentage body fat score by hydrostatic weighing and assigned to one of two groups: non-obese ($< 16\%$) or obese ($\geq 25\%$) (38). Subjects included only untrained men ($VO_{2max} < 45 \text{ ml kg}^{-1} \text{ min}^{-1}$ or < 3 times per week of aerobic or resistance exercise) as classified according to Astrand's table of normative data (5).

Using the variable EPOC, a power calculation (Appendix 2) estimated a sample size of 14 in each group. Since EPOC has a high intra-individual variation, a sample size less than this may be at risk of Type II errors.

Measures:

An overview of the study timeline and testing procedures is illustrated in Figure 1.

BMI

Weight was measured to the nearest 0.1 kg on a beam balance scale. Standing height was measured with a set square to the nearest 0.2 cm. Measurement is expressed as kg/m^2 .

Hydrostatic Weighing

Body density was determined by hydrostatic weighing and percent body fat calculated using the Brozek equation (84). Standard conditions for measurement are detailed in Chapter 1. Fat-free mass was used to express EPOC. In this procedure weight of the subject was recorded in the air, and vital capacity determined from residual volume using the helium dilution technique (79). Water temperature was recorded just prior to the subject entering the tank. A weight belt was placed across the subject's lap to minimize buoyancy in the tank (and was included in the calibration of the chair). The subject was required to eliminate air bubbles from his hair and bathing suit. On the technician's mark, the subject was instructed to take a small comfortable breath prior to being lowered under water and was completely submerged for approximately 10 seconds. A nose clip was used to minimize air loss and the subject was instructed to not exhale during submersion, and to remain as stationary as possible. Upon being raised out of the water, the subject maximally exhaled into a breathing hose to capture lung volume. Eight to ten measurements were taken and the most stable 3 readings were averaged and used in the calculation of body density. This procedure involved weighing the subject underwater, correcting for the residual volume of air trapped in the lungs, and calculating percent body fat from body density.

Maximal oxygen consumption

A graded exercise test to exhaustion ($\dot{V}O_{2\max}$) was administered to determine maximum oxygen consumption. Maximum oxygen consumption ($\dot{V}O_{2\max}$) was used to determine whether the subjects were fit or unfit, according to the definition stated in Chapter 1, and only unfit subjects were included in this investigation. As well, ventilatory threshold (VT) using the $\dot{V}E/\dot{V}CO_2$ method (13) was determined during this session and

used to set the workload of the submaximal exercise session (Exercise/EPOC). The VE/VC_{O_2} threshold was chosen since this threshold often falls closer to 70% VO_{2max} , at which key hormonal responses and possibly a prolonged EPOC are elicited. Work was performed using a Monarch cycle ergometer, heart rate was monitored with a Polar® heart rate monitor (Polar, Port Washington, New York), and ventilatory parameters including VO_2 , VC_{O_2} , VE , and RER were measured via open-circuit spirometry by MedGraphics CPX-D (Minneapolis, Minnesota) or MMC Horizon™ (Beckman, Anaheim CA). To ensure inter-cart validity, the metabolic carts were set-up in parallel with expired gases simultaneously analyzed by both carts. VO_2 at 5 and 10 minutes and at maximum VO_2 were within 10% difference:

Time	Medgraphics		Horizon	
	VO_2 (l/min)	RER	VO_2 (l/min)	RER
5	1.91	0.91	1.80	0.86
10	3.25	1.03	2.92	0.99
VO_{2max} @ 16 min	4.34	1.13	4.21	1.11

Ventilatory measurements on three subjects were performed using the MMC Horizon™ (Beckman, Anaheim CA) due to unavailability of MedGraphics CPX-D (Minneapolis, Minnesota).

VO_{2max} : Subjects were instructed to report to the lab wearing comfortable clothing and to have eaten a small, low fat, low fiber meal at least four hours prior to testing. As well, they were instructed to avoid vigorous exercise for 24 hours prior to the test. Subjects were given a 5-10 minute warm-up on the testing ergometer after which, they were connected to the metabolic cart using a mouthpiece and sampling line. The metabolic cart was used to collect and analyze expired gases continuously. Average expired respiratory gas concentrations were calculated and printed every 15 seconds.

Calibration of the metabolic cart was preformed prior to the start of the test and calibration verification was performed immediately after. Correction for calibration drift was performed when indicated (gas concentration change pre to post test greater than 0.05 units). Heart rate was monitored continuously using a Polar® heart rate monitor (Polar, Port Washington, New York) and recorded every minute during the test.

The exercise test protocol had the subject begin at a resistance of 1.0 kp and a pedal speed of 60-80 rpm. The resistance was increased 0.5 kp every 2 minutes until VT and then increased at 1 minute intervals. Subjects were instructed to maintain the 60-80 rpm pedal speed. The test continued until volitional exhaustion. $\dot{V}O_{2\max}$ was considered to be attained if 2 of the following criteria were met: 1) volitional exhaustion, 2) $RER \geq 1.1$, 3) maximum age-predicted heart rate ($220 - \text{age}$) is attained, 4) peak and plateau in $\dot{V}O_2$ despite increased workload (40).

Diet Record

Energy intake was determined using a 3 day prospective diet record of daily food intake including all fluids. The 3 day period occurred over consecutive days and included a Saturday or Sunday. Total calories were calculated as well as determination of caloric intake derived from carbohydrates, fats and protein. This was analyzed using The Food Processor nutritional analysis software (ESHA, Portland, Oregon). Subjects were given instructions and a sample dietary record to assist them and were requested to be as explicit as possible in documenting information. The diet record was used to help establish whether subjects maintained their regular mixed diets and caloric intake prior to each experiment trial. On the day of the **Baseline** and **Exercise/EPOC** protocol, subjects were asked to consume meals that were similar or identical to those recorded in the diet record. One day of the diet record was photocopied and the subject was asked to follow it for his meal plan prior to the **Baseline** and **Exercise/EPOC** sessions.

Exercise bout:

Subjects exercised continuously on a cycle ergometer at their individual ventilatory threshold for 30 minutes. The power output corresponding to ventilatory threshold was determined from the $\dot{V}O_{2\max}$ test according to the $\dot{V}E/\dot{V}CO_2$ method and was used to set the intensity of exercise. An intensity level set at VT was chosen since key hormonal responses and possibly a prolonged EPOC are elicited at higher intensity exercise ($> 60\% \dot{V}O_{2\max}$ and/or > 20 min). A $\dot{V}O_{2\max}$ of 60-70% falls close to where VT is expected to occur for untrained subjects. As stated earlier, cortisol progressively increases according to intensity and duration and there may exist a threshold of intensity at 60-70% $\dot{V}O_{2\max}$ and of duration greater than 20 minutes whereby cortisol levels increase significantly in the post exercise period. This intensity of exercise falls close to where anaerobic threshold is expected to occur for untrained subjects. Growth hormone also demonstrates a more pronounced increase with greater intensities and durations of exercise. The increase in both hormones, however, is not immediate but occurs gradually with time (88). And, as suggested by Frey et al. (43), variability of reported EPOC results maybe decreased by setting workloads relative to each individual's anaerobic threshold.

The exercise bout commenced for all subjects starting at 2:00 to 3:00 pm. Power output, heart rate, RPE, and tympanic temperature were recorded. Tympanic temperature was measured using the Thermoscan® infrared thermometer (Braun, USA) by inserting the head of the thermometer into the ear canal of the subject. Heart rate was monitored using a Polar® heart rate monitor.

Excess post-exercise oxygen consumption (EPOC):

Oxygen consumption was measured during the recovery period following the exercise challenge (Exercise/EPOC). Also oxygen consumption was assessed on an alternate day at the same time and under the same conditions but without the exercise intervention to serve as baseline data (Baseline). For both sessions, oxygen consumption was measured continuously by connecting the subject to the metabolic cart (MedGraphics CPXD) using a mouthpiece for the 30 minutes, followed by interval gas collection using a canopy for the next 80 minutes. Heart rate and tympanic temperature were also monitored over these sessions. EPOC magnitude was calculated by summing excess $\dot{V}O_2$ during the recovery period. The cessation of EPOC was taken as two or more consecutive minute values of oxygen consumption at or below the baseline resting $\dot{V}O_2$ (33). Based on the literature, the exercise protocol was not anticipated to elicit EPOC greater than two hours.

Hormone and Lactate Levels:

An intravenous catheter was inserted prior to the start of the Baseline and Exercise/EPOC tests and venous blood samples collected before (-30 min), during (0,30 min), and after exercise (50, 70, 90, 110, 130 min) as shown in Figure 2. A 3 ml sample of blood at each of the time points was collected. The total amount of blood collected from each subject was 27 ml for each session. With each blood sample, 0.5 ml was mixed with 2 ml of perchloric acid, chilled 5 minutes on ice, centrifuged for 10 minutes, and the supernatant frozen for lactate determination. The remaining blood was allowed to clot at room temperature for 45 minutes. The blood was centrifuged (10 minutes at 2500 rpm) and the serum collected was stored at -80°C and analyzed at a later time. Hormonal assays were performed on serum samples in duplicate using appropriate radioimmunoassay kits for GH, cortisol, T_3 , and T_4 .

All hormones were assayed using the GammaCoat™ [¹²⁵I] commercially prepared radioimmunoassay kits. To minimize interassay variability, samples from the same subject were analyzed in duplicate on the same testing day for each hormone. Procedures used were obtained from the GammaCoat™ [¹²⁵I] RIA kit instruction manuals. Serum controls and the number of computer iterations required to plot the standard curve for the assay were used to assess the validity of the assays. The precision of the assays was measured by computer generated coefficient of variation (CV). The CV's were within the acceptable range of 6-15% (26).

Data Analysis:

Data were analyzed using inferential statistical procedures. Student's T-test was used to compare subject characteristics and $\dot{V}O_2$ response during Baseline and Exercise/EPOC between groups. One-way ANOVA was used to analyze caloric intake (energy and macronutrient composition). Repeated Measures ANOVA was used to analyze “**Between Group**” differences for lean and obese groups (main effect) for tympanic temperature, lactate, heart rate, RER, and hormones. This analysis was performed comparing lean versus obese during Baseline to assess differences between groups at rest. A separate Repeated Measures ANOVA was applied to the Exercise/EPOC sessions to compare lean versus obese response to the exercise intervention. This was followed by 1-way ANOVA when significant difference was found.

Repeated Measures ANOVA was also used to analyze differences between the Baseline and Exercise/EPOC session (“**Between Session**” comparison) for the lean and obese groups separately. If significance was observed, a one-way analysis of variance (ANOVA) was applied. Non-significant differences between Baseline and

Exercise/EPOC were the time points used to identify the recovery of each variable assessed. For all statistical procedures, the significance level was set at $p < 0.05$.

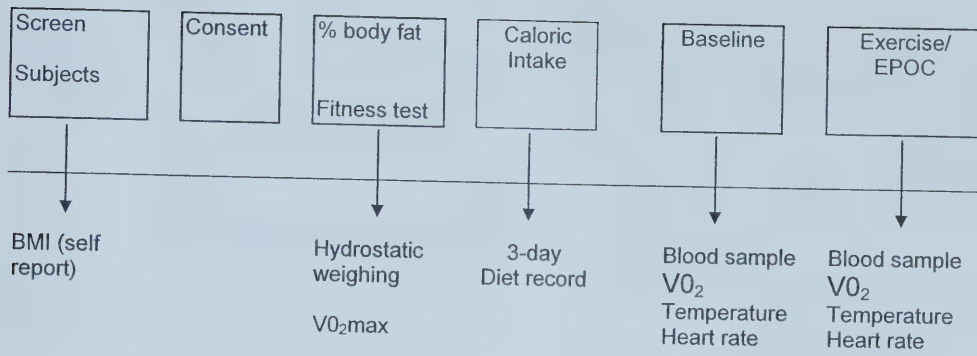
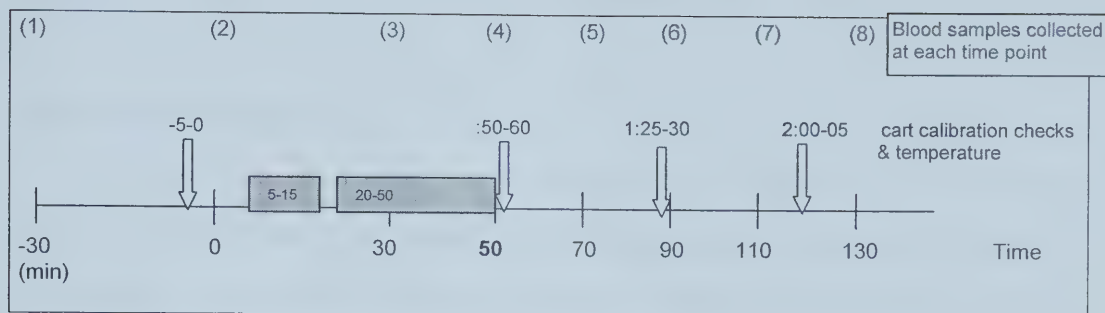


Figure 1: Overview of procedures

Baseline



Exercise/EPOC

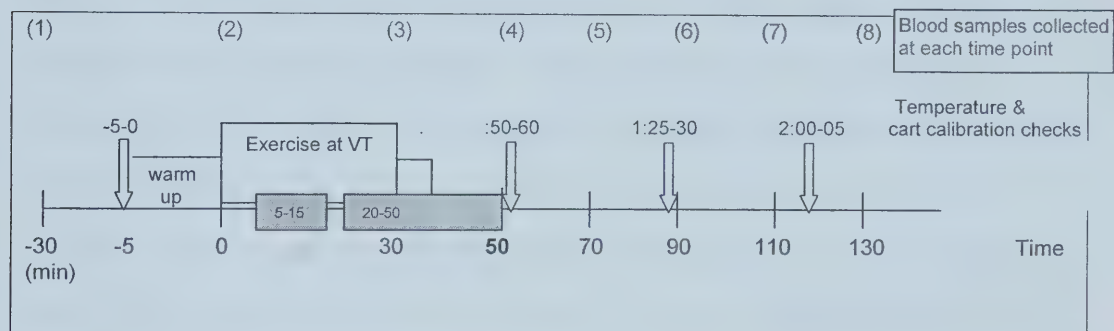


Figure 2. Baseline (160 minutes rest) and Exercise/EPOC (30 min rest, 30 min exercise at ventilatory threshold on a stationary bike, 5 min cool-down, 100 min recovery) sessions. Blood samples (3 ml) were drawn at 8 sampling points.

Continuous gas collection from time 20 to 50 minutes (via mouthpiece). Interval gas collection from time 50 to 130 minutes (via canopy).

Chapter 4: Results

Subject Characteristics:

Subjects were recruited with poster advertisement as well as through a television news broadcast. From approximately 50 respondents, 30 were screened (BMI and delimitations) and invited to participate in the study. A "Participant Information Letter" was handed out and informed consent was administered. Eighteen subjects met the inclusion criteria for percentage body fat and fitness level. Two subjects declined to participate and two subjects dropped out midway through the study. One subject completed the study but his data were excluded because his serum samples appeared "cloudy". Turbid, cloudy, or milky serum (lipemic serum) may be produced by the presence of fatty substances (lipids) in the blood. This may be the result of a recent meal or high fasting cholesterol levels. Bacterial contamination may also cause cloudy serum. Data from 13 subjects were analyzed.

There were 7 subjects in the obese group and 6 subjects in the lean group. Table I presents mean $\pm$ standard deviation (SD) data summarizing the characteristics of the obese and lean groups. The groups were similar with respect to age and height. Compared to the lean, the obese group had a significantly greater proportion of body fat (obese= $32.2 \pm 6\%$; lean= $15.4 \pm 1\%$ fat), and larger total body mass (obese= 103.3 ± 11.4 kg; lean= 66.3 ± 7.4 kg), fat mass (obese= 33.5 ± 8.3 ; lean= 10.2 ± 1.4 kg) and fat-free mass (obese= 69.8 ± 7.2 kg; lean= 56.1 ± 6.1 kg). Absolute $\dot{V}O_{2\max}$ was higher in the obese than lean group (obese= 3.2 ± 0.5 l/min; lean= 2.7 ± 0.3 l/min), relative $\dot{V}O_{2\max}$ per kg was lower (obese= 31 ± 6.1 ml/kg/min; lean= 41.8 ± 4.4 ml/kg/min), and relative $\dot{V}O_{2\max}$ per fat-free mass (FFM) was similar (obese= 46.2 ± 5.7 ml/kg FFM/min; lean= 48.7 ± 4.6 ml/kg

FFM/min). There was no significant difference in ventilation threshold (obese= $67 \pm 5.3\%$ $\dot{V}O_{2\max}$; lean= $68 \pm 8.2\%$ $\dot{V}O_{2\max}$).

Oxygen Consumption

Table II presents oxygen consumption (mean $\pm$ SD) during Baseline and EPOC sessions and is reported in absolute (l/min), relative to total body weight (ml/kg/min), and relative to fat-free mass (ml/kg FFM/min) values. One subject from the lean group was not included in the calculation due to a problem with the metabolic cart during testing. Total magnitude of $\dot{V}O_2$ during the 2.5 hour Baseline session in the obese group was: 23.3 ± 1.9 l/min, 226.6 ± 11.2 ml/kg/min, and 335.8 ± 28.3 ml/kg FFM/min. Baseline $\dot{V}O_2$ in the lean group was: 18.1 ± 0.9 l/min, 276.5 ± 40.1 ml/kg/min, and 326.0 ± 46.8 ml/kg FFM/min. Resting oxygen consumption in the obese group was greater when expressed in absolute values ($p < 0.05$), lower when expressed relative to body weight ($p < 0.05$), and similar when the comparison is made relative to fat-free mass.

In response to exercise, EPOC magnitude in the lean group (4.2 ± 0.6 l/min; 64.5 ± 13.2 ml/kg/min; 76.0 ± 15.0 ml/kg FFM/min) was greater than the obese group (3.0 ± 1.3 l/min; 29.3 ± 13.7 ml/kg/min; 43.9 ± 20.2 ml/kg FFM/min) ($p < 0.05$). Duration of EPOC was also significantly longer in the lean group (46.8 ± 6.7 minutes) than in the obese (27.9 ± 14.8 minutes). Although the half-time ($T_{1/2}$) for the rate of decrease in $\dot{V}O_2$ for the “rapid” component of EPOC was slightly faster for the lean group, the duration of the rapid component was 5 minutes for both groups and the magnitude of oxygen consumed during this period was not different (lean= 756 ± 109.3 ml/min; obese= 705 ± 187.8 ml/min).

Caloric Intake:

The subjects in each group completed a 3-day journal of caloric intake. All but 2 subjects, one from each group returned their dietary intake journal. Table III presents averaged values for total calories consumed and proportion and gram weight of protein, carbohydrates, and fat. There were no group differences when analyzed by ANOVA. Although not statistically significant, the lean group consumed more calories than the obese group (2711 ± 417 kcal vs. 2240 ± 726 kcal).

Tympanic temperature, lactate, and heart rate:

Lean and obese group responses (mean $\pm$ SD) for tympanic temperature, lactate, heart rate, and RER are presented in Table IV. Between session (Baseline vs. Exercise/EPOC) analysis was performed on each group separately to determine the duration that each variable remained elevated above resting levels (Figure 10). Between group (lean vs. obese) analysis was performed to determine whether there was a difference between lean and obese group response.

The exercise stimulus did not elicit any significant changes in tympanic temperature for either between session or between group comparisons. Not all time points were measured on each subject when delays with the calibration of the metabolic cart occurred. A minimum of 3 measurements were used for the Baseline session and a minimum of 6 measurements were used for the Exercise/EPOC session.

Blood lactate during Exercise/EPOC in the lean group remained above resting levels until Time70-90 and in the obese group until Time50-70. There was no difference between groups at rest. Exercise had a similar effect between lean and obese groups during Exercise/EPOC, including peak response after 30 minutes of exercise (Time30), which was 6.0 ± 2.9 mM in the lean and 4.6 ± 1.8 mM in the obese ($p=0.284$).

Heart rate response during Exercise/EPOC, in the obese group, persisted above resting levels throughout the entire recovery period, while, in the lean group, returned to resting levels by Time70. Between group comparison during Exercise/EPOC, however, (Figure 4) revealed no difference between lean and obese groups response ($p=0.55$) including peak heart rate at Time30 (lean= 155 ± 21 beats/min; obese= 159 ± 13 beats/min). There was also no difference between groups at rest.

RER:

RER was monitored continuously and each 5 minute interval was averaged (Table V). RER during the Exercise/EPOC session, compared to Baseline, was lower in the lean group throughout the recovery period and was statistically significant at all time points except Time86-95 (Figure 5b). Although RER in the obese group was lower during Exercise/EPOC compared to Baseline from Time60-90, this was not statistically different (Figure 5a).

There was no difference in RER between groups at rest ($p=0.364$). RER over the Baseline session averaged 0.78 ± 0.04 for the obese group and 0.80 ± 0.03 for the lean group. In response to exercise and during the recovery period, RER in the obese group remained significantly higher than the lean group (Figure 5c). Investigation of 5 minute time segments revealed significant differences between lean and obese during the following recovery times: Time60-65 ($p=0.03$), Time65-70 ($p=0.018$), Time70-75 ($p=0.003$), Time75-80 ($p=0.003$), Time115-120 ($p=0.054$), and Time125-130 ($p=0.024$). This accounts for 30 out of 100 minutes (30%) of the recovery period.

Hormones:

Table VI presents the mean $\pm$ SD concentrations for cortisol, growth hormone (GH), T_3 , and T_4 at each of the 8 sampling points during the Baseline and

Exercise/EPOC sessions. These results are illustrated in Figures 6-9. Technical difficulties during one blood draw prevented blood collection and subsequent hormone analysis for that particular sampling point.

Growth Hormone:

In the lean group, GH levels during Exercise/EPOC remained significantly elevated above Baseline throughout the entire recovery period (Time30-130), while in the obese group, GH returned to non-significant values by Time70. Growth Hormone (GH) concentrations between the 2 groups were similar at rest ($p=0.162$). During the Exercise/EPOC session, the lean group demonstrated greater GH concentrations compared to the obese group ($p=0.005$). Peak GH response to exercise was 44.0 ± 22.2 ng/ml and 13.4 ± 12.2 ng/ml and for the lean and obese, respectively ($p<0.05$). Figure 7 illustrates the GH response during Baseline and Exercise/EPOC.

Cortisol:

In the lean group, cortisol levels during Exercise/EPOC were significantly elevated above Baseline levels from Time30-110, and in the obese group, remained significantly elevated at Time130. Between group analysis of cortisol concentrations revealed similar resting levels in the obese and lean ($p=0.091$), although, as shown in Figure 6, the obese appear to have higher resting levels (obese= 9.7 ± 1.9 µg/dl; lean= 5.8 ± 1.7 µg/dl) this was not significant. The higher basal condition in the obese group was influenced by an exaggerated response of one subject who fainted during the initial blood draw. Variation within this group was, therefore, comparably large and contributed to a significant Levene's statistic for equality of variance between groups. Nonetheless, significance was unaffected since no significant difference was found despite the increased possibility of detecting one due to the variance. Throughout the

Exercise/EPOC session, cortisol levels in the obese were greater than the lean ($p=0.02$). Peak concentration occurred at Time30 and was 25.5 ± 7.7 $\mu\text{g/dl}$ in the obese and 13.1 ± 5.3 $\mu\text{g/dl}$ in the lean ($p<0.05$). Figure 6 illustrates the response of the lean and obese groups during the Exercise/EPOC session.

T₃ and T₄:

T₃ concentrations during Exercise/EPOC did not significantly increase above Baseline concentrations in the lean or obese groups. Peak response in the obese group was 1.8 ± 0.2 μM and the lean was 1.3 ± 0.2 μM at Time30 (Figure 8). Between group analysis revealed significantly higher T₃ concentrations in the obese than the lean subjects during both Baseline and Exercise/EPOC sessions. The pre-existing difference (0.4 μM) between groups at rest was preserved during exercise and, hence, T₃ levels in the obese remained higher than the lean during Exercise/EPOC ($p<0.05$).

T₄ concentrations during Exercise/EPOC were not significantly different from Baseline in obese or lean groups. Peak response in the obese group was 8.9 ± 1.2 $\mu\text{g/dl}$ and the lean group was 9.7 ± 2.7 $\mu\text{g/dl}$ at Time30 (Figure 9). Between group analysis also revealed no differences between lean and obese groups during either Baseline ($p=0.549$) or Exercise/EPOC ($p=0.421$) sessions.

Table I. Characteristics of lean and obese subjects (mean±SD).

	Obese (n=7)	Lean (n=6)
Age (yrs)	36.1±3.4	34.5±2.6
Height (cm)	179.9±3.7	175.1±10.1
Weight (kg)	103.3±11.4*	66.3±7.4
Fat mass (kg)	33.5±8.3*	10.2±1.4
FFM (kg)	69.8±7.2*	56.1±6.1
Body fat (%)	32.2±5.7*	15.4±1
V _{O₂max} (l/min)	3.2±0.5*	2.7±0.3
V _{O₂max} (ml/kg/min)	31.3±6.1*	41.8±4.4
V _{O₂max} (ml/kg FFM/min)	46.2±5.7	48.7±4.6
V _{O₂max} at VT (%)	67.4±5.3	68±8.2
PO at VT (watts)	154±35	155±34

* Significantly different from lean at p<0.05.

FFM = fat-free mass

VT = ventilatory threshold for VE/VCO₂

PO = power output

Table II: Oxygen consumption (AUC±SD) during Baseline¹ and EPOC² conditions in lean and obese subjects

	Obese (n=7)	Lean (n=5) ³
Baseline¹:		
Absolute (l)	5.5±2.8 ^A	7.9±1.9 ^B
Relative (ml/kg/min)	54.1±29.4 ^{A*}	119.7±30.4 ^B
Relative (ml/kg FFM/min)	80.1±41.7 ^{A*}	141.2±35.9 ^B
EPOC²:		
Absolute (l)	8.5±4.0*	12.1±1.9
Relative (ml/kg/min)	83.6±42.1*	184.2±36.1
Relative (ml/kg FFM/min)	124.3±59.8*	217.1±41.8
Net:		
Absolute (l)	3.0±1.3*	4.2±0.6
Relative (ml/kg/min)	29.3±13.7*	64.5±13.2
Relative (ml/kg FFM/min)	43.9±20.2*	76.0±15.0
Duration of EPOC:	27.9±14.8*	46.8±6.7
Rapid component of EPOC:		
V _O ₂ (ml/min)	705±187.8	756±109.3
T _{1/2} (sec)	109	73.2
Duration (min)	5	5
Rate:		
Baseline (ml/min)	307±25.6*	238±11.5
(ml/kg/min)	3.0±0.1*	3.6±0.5
(ml/kg FFM/min)	4.4±0.4	4.3±0.6
EPOC: (ml/min)	480±97*	371±27
(ml/kg/min)	4.6±0.7*	5.7±0.9
(ml/kg FFM/min)	6.9±1.5	6.7±1.0
Net: (ml/min)	174±79	133±31
(ml/kg/min)	1.6±0.7	2.0±0.6
(ml/kg FFM/min)	2.5±1.2	2.4±0.7

* Significantly different from lean at p<0.05.

AUC = area under the curve.

FFM = fat-free mass

¹Baseline is calculated from Time35 to the end of EPOC (determined from the Exercise/EPOC session). Therefore calculation involves a different duration between groups and includes a calibration break.

^ACalculated for 28 minutes from Time35-58

^BCalculated for 47 minutes from Time35-77

²EPOC is calculated during the Exercise/EPOC session; from Time35 to the end of EPOC: until V_O₂ is equal to or below values of the Baseline session.

³Data from 1 subject was not included due to a problem with the metabolic cart.

Table III. Daily caloric intake (mean $\pm$ SD) averaged from 3-day energy intake record in lean and obese subjects

	Obese (n=6) ^A	Lean (n=5) ^A
Protein (g)	84 $\pm$ 20	110 $\pm$ 20
Protein (%)	15 $\pm$ 4	16 $\pm$ 1
CHO (g)	296 $\pm$ 106	335 $\pm$ 25
CHO (%)	52 $\pm$ 2	50 $\pm$ 10
Fat (g)	81 $\pm$ 26	93 $\pm$ 40
Fat (%)	32 $\pm$ 3	29 $\pm$ 9
Calories (kcal)	2240 $\pm$ 726	2711 $\pm$ 417

CHO = carbohydrate

^AOne subject from each group did not return diet record.

Table IV: Lean and obese group response (mean $\pm$ SD) during Baseline (A) and Exercise/EPOC (B) for tympanic temperature, heart rate, and lactate.

A) Baseline

	Time (min)							
	-30	0	30	50	70	90	110	130
Obese								
Temperature	37 $\pm$ 0.7	36 $\pm$ 0.8	36 $\pm$ 0.6	36 $\pm$ 1.0	–	36 $\pm$ 0.7	36 $\pm$ 0.5	36 $\pm$ 0.9
HR	70 $\pm$ 7.3	66 $\pm$ 8.2	67 $\pm$ 8.1	64 $\pm$ 5.9	61 $\pm$ 6.1	60 $\pm$ 9.1	62 $\pm$ 6.3	61 $\pm$ 4.8
Lactate	0.4 $\pm$ 0.3	0.5 $\pm$ 0.2	0.3 $\pm$ 0.3	0.3 $\pm$ 0.3	0.4 $\pm$ 0.3	0.4 $\pm$ 0.3	0.5 $\pm$ 0.3	0.4 $\pm$ 0.4
Lean								
Temperature	37 $\pm$ 0.2	36 $\pm$ 0.5	36 $\pm$ 0.5	36 $\pm$ 0.3	–	36 $\pm$ 0.3	36 $\pm$ 0.3	36 $\pm$ 0.5
HR	70 $\pm$ 6.5	72 $\pm$ 5.9	65 $\pm$ 9.4	66 $\pm$ 12.6	60 $\pm$ 8.1	65 $\pm$ 6.0	63 $\pm$ 9.1	61 $\pm$ 7.3
Lactate	0.6 $\pm$ 0.4	0.4 $\pm$ 0.3	0.6 $\pm$ 0.5	0.3 $\pm$ 0.1	0.3 $\pm$ 0.2	0.2 $\pm$ 0.2	0.4 $\pm$ 0.1	0.4 $\pm$ 0.4

B) Exercise/EPOC

	Time (min)							
	-30	0	30	50	70	90	110	130
Obese								
Temperature	37 $\pm$ 0.6	37 $\pm$ 0.3	36 $\pm$ 0.7	36 $\pm$ 0.7	–	37 $\pm$ 0.7	36 $\pm$ 0.5	36 $\pm$ 0.9
HR	74 $\pm$ 7.6	112 $\pm$ 14.6	157 $\pm$ 13.6	95 $\pm$ 10.6	78 $\pm$ 6.8	76 $\pm$ 9.9	73 $\pm$ 6.0	71 $\pm$ 6.1
Lactate	0.4 $\pm$ 0.2	1.2 $\pm$ 0.8	4.6 $\pm$ 1.8	1.6 $\pm$ 0.8	0.9 $\pm$ 0.4	0.6 $\pm$ 0.4	0.5 $\pm$ 0.3	0.5 $\pm$ 0.2
Lean								
Temperature	36 $\pm$ 0.2	36 $\pm$ 0.4	37 $\pm$ 1.2	36 $\pm$ 0.6	–	36 $\pm$ 0.3	36 $\pm$ 0.3	36 $\pm$ 0.3
HR	71 $\pm$ 13.5	114 $\pm$ 12.3	154 $\pm$ 18.8	87 $\pm$ 14.7	72 $\pm$ 12.4	73 $\pm$ 14.4	71 $\pm$ 15.3	75 $\pm$ 14.4
Lactate	0.5 $\pm$ 0.4	2.2 $\pm$ 1.4	6.0 $\pm$ 2.9	2.1 $\pm$ 1.8	1.3 $\pm$ 1.1	1.0 $\pm$ 0.6	0.7 $\pm$ 0.4	0.6 $\pm$ 0.3

HR = heart rate

Table V: Lean and obese group response (mean±SD) for RER¹ during Baseline and Exercise/EPOC.

Time (min)	Baseline		Exercise/EPOC	
	Obese	Lean	Obese	Lean
35	0.79±0.04	0.80±0.03	0.87±0.11	0.92±0.07
40	0.80±0.04	0.80±0.04	0.83±0.11	0.88±0.13
45	0.80±0.06	0.81±0.05	0.83±0.13	0.77±0.07
50	0.78±0.05	0.76±0.07	0.79±0.09	0.78±0.13
65	0.80±0.04	0.81±0.03	0.73±0.02*	0.65±0.05
70	0.83±0.07	0.83±0.05	0.75±0.04*	0.65±0.08
75	0.81±0.06	0.80±0.05	0.76±0.05*	0.65±0.05
80	0.79±0.03	0.80±0.06	0.75±0.04*	0.66±0.03
85	0.79±0.1	0.80±0.08	0.72±0.07	0.67±0.04
95	0.77±0.09	0.81±0.07	0.78±0.1	0.76±0.02
100	0.76±0.09	0.79±0.07	0.78±0.09	0.70±0.03
105	0.79±0.1	0.80±0.06	0.77±0.12	0.70±0.03
110	0.81±0.07	0.82±0.06	0.79±0.14	0.70±0.05
115	0.79±0.05	0.82±0.04	0.79±0.13*	0.70±0.06
120	0.74±0.05	0.82±0.07	0.84±0.06*	0.71±0.06
125	0.73±0.06	0.83±0.08	0.87±0.06	0.74±0.03
130	—	—	0.84±0.08*	0.74±0.01

*Significantly different from lean at $p<0.05$.

¹From Time35 through Time130, 5 minute intervals were averaged.

Table VI. Serum levels (mean±SD) of cortisol (µg/dl), GH (ng/ml), T₃ (ng/dl), and T₄ (µg/dl) in obese and lean subjects at each time point (min) during Baseline (A) (160 min rest) and Exercise/EPOC (B) (30 min rest, 30 min exercise, 100 min recovery) conditions.

A) Baseline

	Time (min)							
	-30	0	30	50	70	90	110	130
Obese								
Cortisol	9.4±5.0	12.0±7.3	12.0±8.0	11.1±7.5	9.7±5.3	4.0±4.5	7.3±4.1	7.4±4.4
GH	0.2±0.2	1.9±3.3	1.0±1.3	0.8±1.3	0.5±0.7	0.3±0.5	0.5±0.9	0.7±1.2
T ₃	1.6±0.3*	1.5±0.2*	1.5±0.2*	1.5±0.2*	1.4±0.3*	1.4±0.3*	1.3±0.2*	1.4±0.1*
T ₄	8.4±1.1	7.5±0.7	7.9±1.2	7.9±1.1	7.6±0.8	7.6±1.1	7.6±1.1	7.6±1.0
Lean								
Cortisol	8.1±2.2	7.9±1.2	6.6±1.5	6.2±1.6	5.2±1.2	4.3±1.3	3.9±1.6	4.0±1.4
GH	6.8±12.8	9.8±14.3	4.5±6.2	2.7±3.8	1.7±2.0	0.9±1.1	0.5±0.7	0.3±0.4
T ₃	1.3±0.2	1.1±0.1	1.1±0.2	1.1±0.2	1.0±0.1	1.0±0.1	1.0±0.1	1.1±0.2
T ₄	9.3±3.4	7.9±1.4	8.2±1.1	8.6±2.2	7.9±1.2	8.5±1.8	7.6±2.0	7.9±1.3

B) Exercise/EPOC

	Time (min)							
	-30	0	30	50	70	90	110	130
Obese								
Cortisol	9.9±3.2	10.5±5.0	25.5±7.7**	26.2±7.4**	20.4±6.5**	16.6±6.6**	14.3±4.9**	12.8±3.6**
GH	0.4±0.8	1.2±1.8**	13.4±12.2**	4.7±3.4**	1.9±1.3**	0.8±0.6**	0.4±0.3**	0.3±0.3**
T ₃	1.7±0.2**	1.7±0.1**	1.8±0.2**	1.6±0.2**	1.3±0.2**	1.4±0.2**	1.4±0.1**	1.4±0.2**
T ₄	8.1±1.2	8.7±0.9	8.9±1.2	7.7±1.3	7.7±1.0	7.5±1.1	7.6±1.0	7.9±1.2
Lean								
Cortisol	8.1±2.5	7.1±1.9	13.1±5.3	13.2±6.2	10.9±5.8	9.3±5.4	8.0±3.9	7.0±3.3
GH	7.2±8.8	12.6±10.8	43.9±22.2	18.6±12.1	8.5±5.3	4.2±3.1	2.2±1.4	1.3±0.8
T ₃	1.3±0.1	1.2±0.1	1.3±0.2	1.2±0.1	1.0±0.1	1.0±0.3	1.0±0.1	0.9±0.1
T ₄	9.4±2.3	8.7±1.7	9.7±2.7	9.1±1.6	9.1±1.8	8.7±1.9	8.7±2.2	9.1±1.8

* Obese vs. lean at Baseline: significantly different from lean at p<0.05.

** Obese vs. lean at Exercise/EPOC: significantly different from lean at p<0.05.

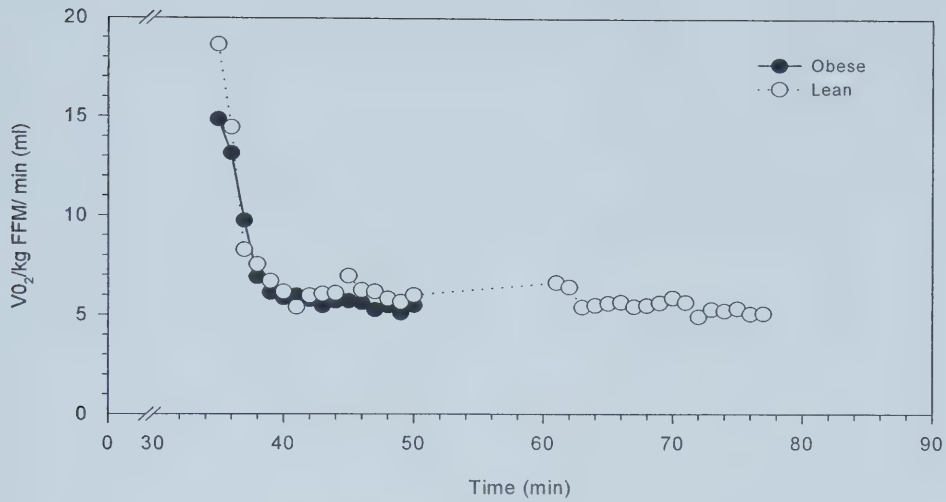


Figure 3. Time plot of EPOC for obese (n=7, black circle) and lean (n=5, white circle) subjects following 30 minutes of exercise at ventilatory threshold (+ 5min cool-down) on a stationary bike. Error bars (SD) have been omitted.

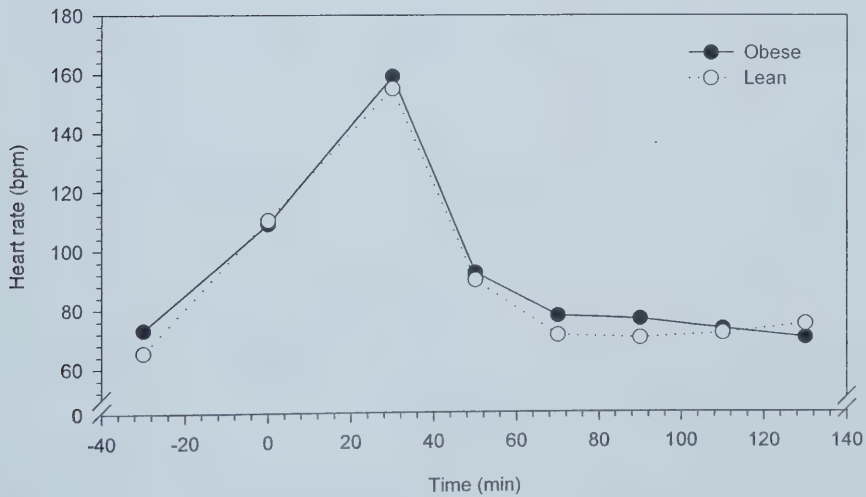


Figure 4. Heart rate response of obese (n=7, black circle) and lean (n=6, white circle) subjects during the Exercise/EPOC session.

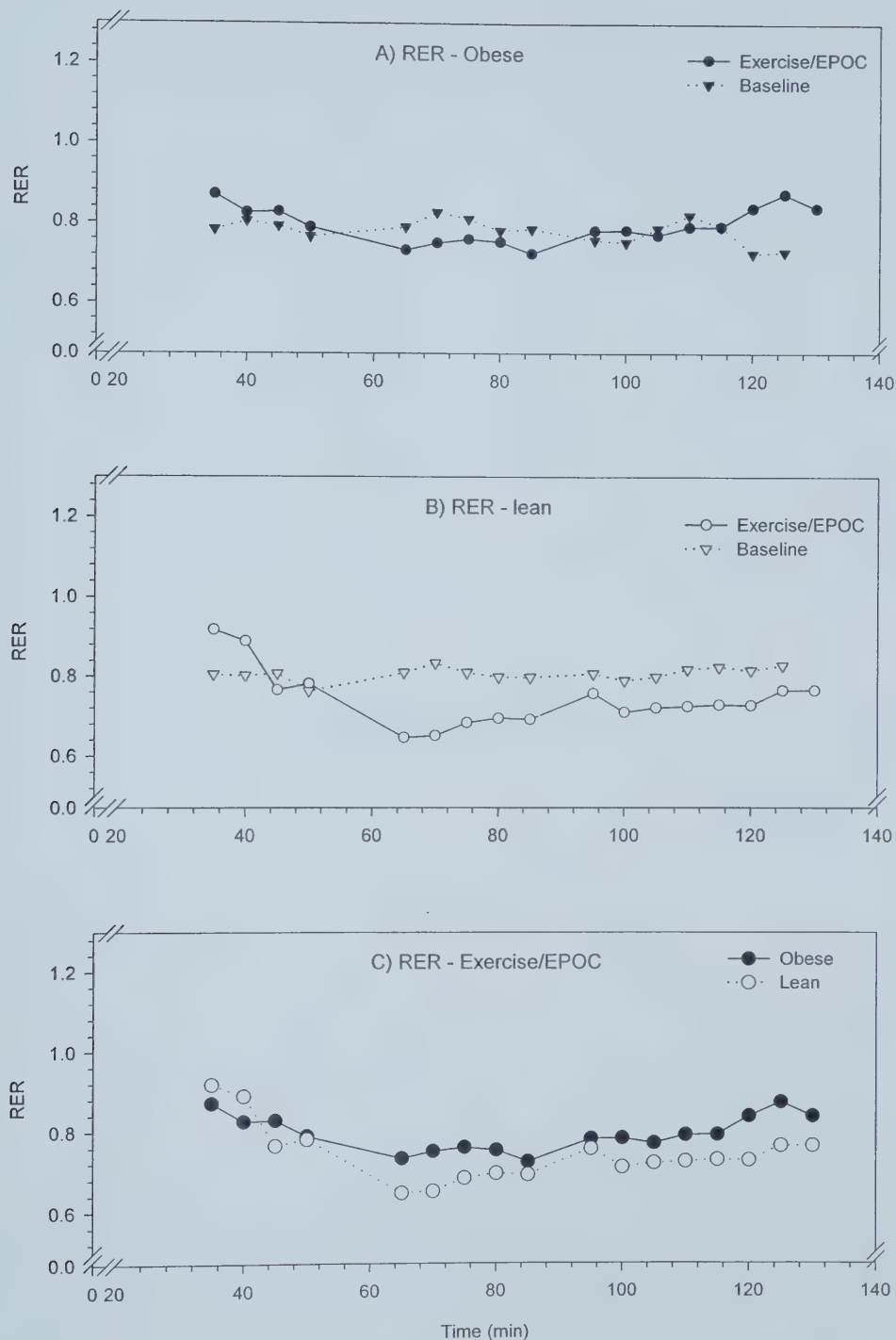


Figure 5. Time plots of mean RER value in obese ($n=7$, black symbol) (A) and in lean ($n=6$, white symbol) (B) subjects during Baseline (160 min rest) and Exercise/EPOC (30 min rest, 30 min exercise, 100 min recovery) conditions. Comparison of RER response in obese and lean subjects during Exercise/EPOC (C). Error bars (SD) have been omitted.

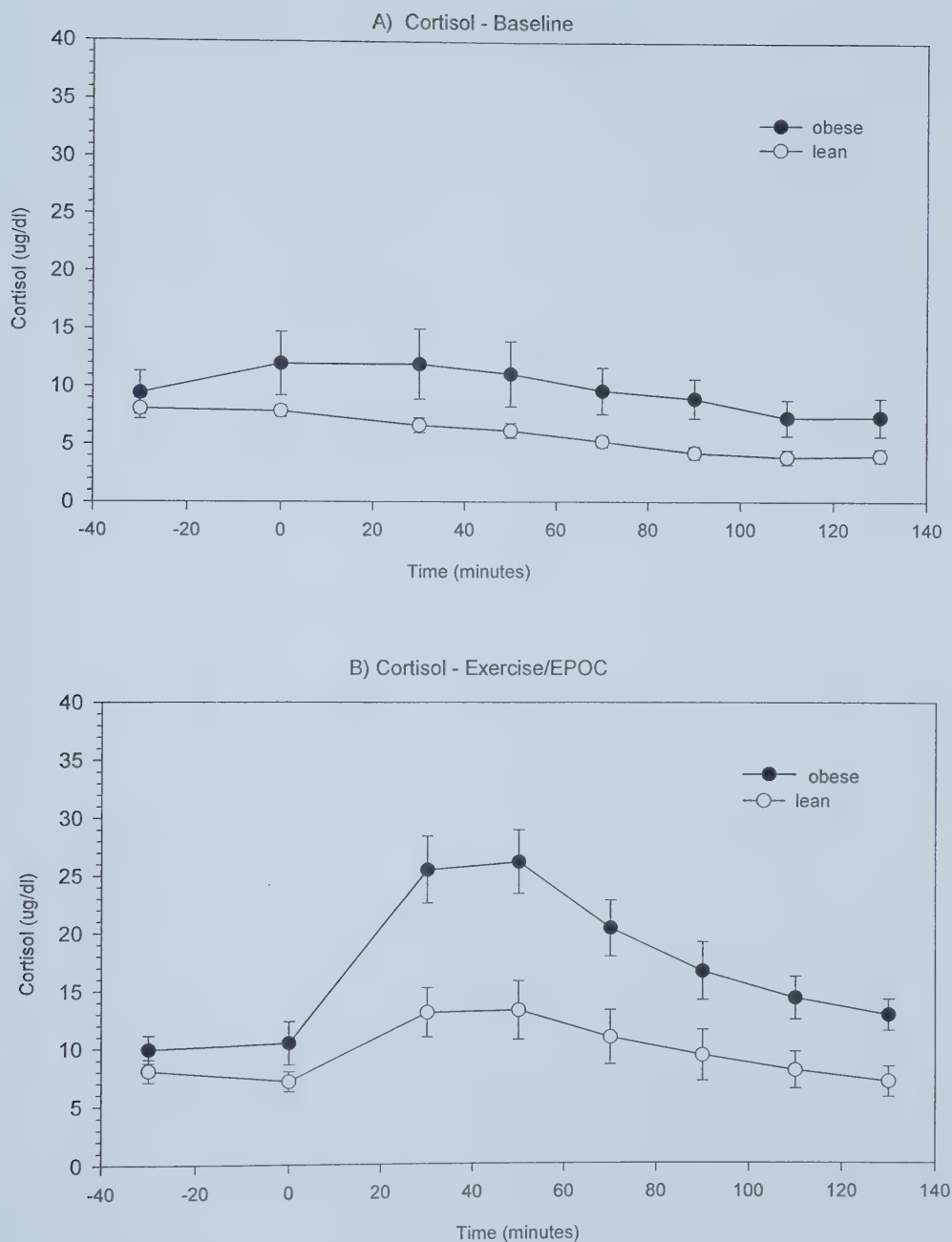


Figure 6. Serum cortisol levels during the Baseline (160 min rest) (A) and during Exercise/EPOC (30 min rest, 30 min exercise at VT, 100 min recovery) (B) conditions in 7 obese (black circles) and 6 lean (white circles) subjects. Values are expressed as mean $\pm$ SD.

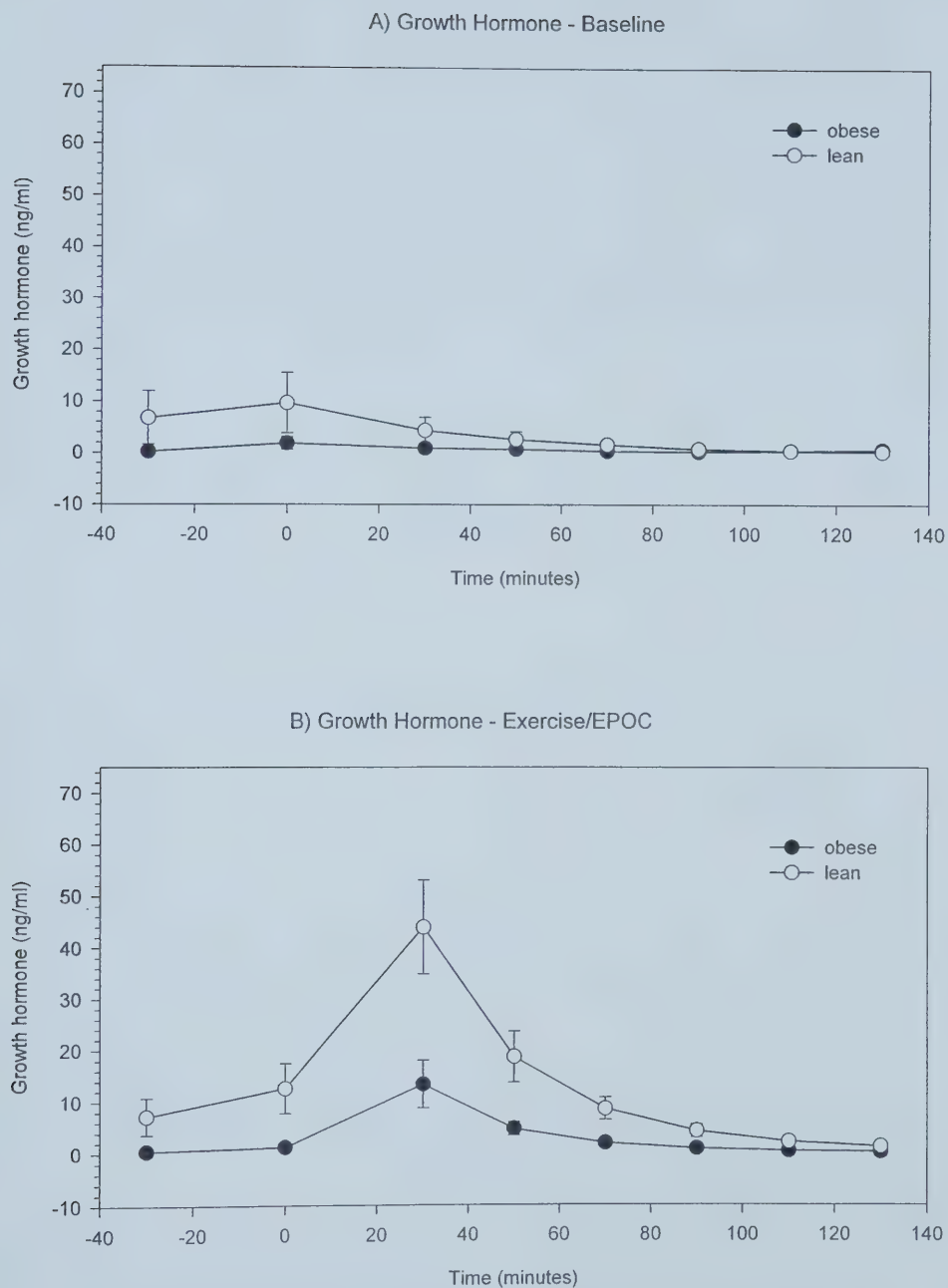


Figure 7. Serum GH levels during the Baseline (160 min rest) (A) and during Exercise/EPOC (30 min rest, 30 min exercise at VT, 100 min recovery) (B) conditions in 7 obese (black circle) and 6 lean (white circle) subjects. Values are expressed as mean $\pm$ SD.

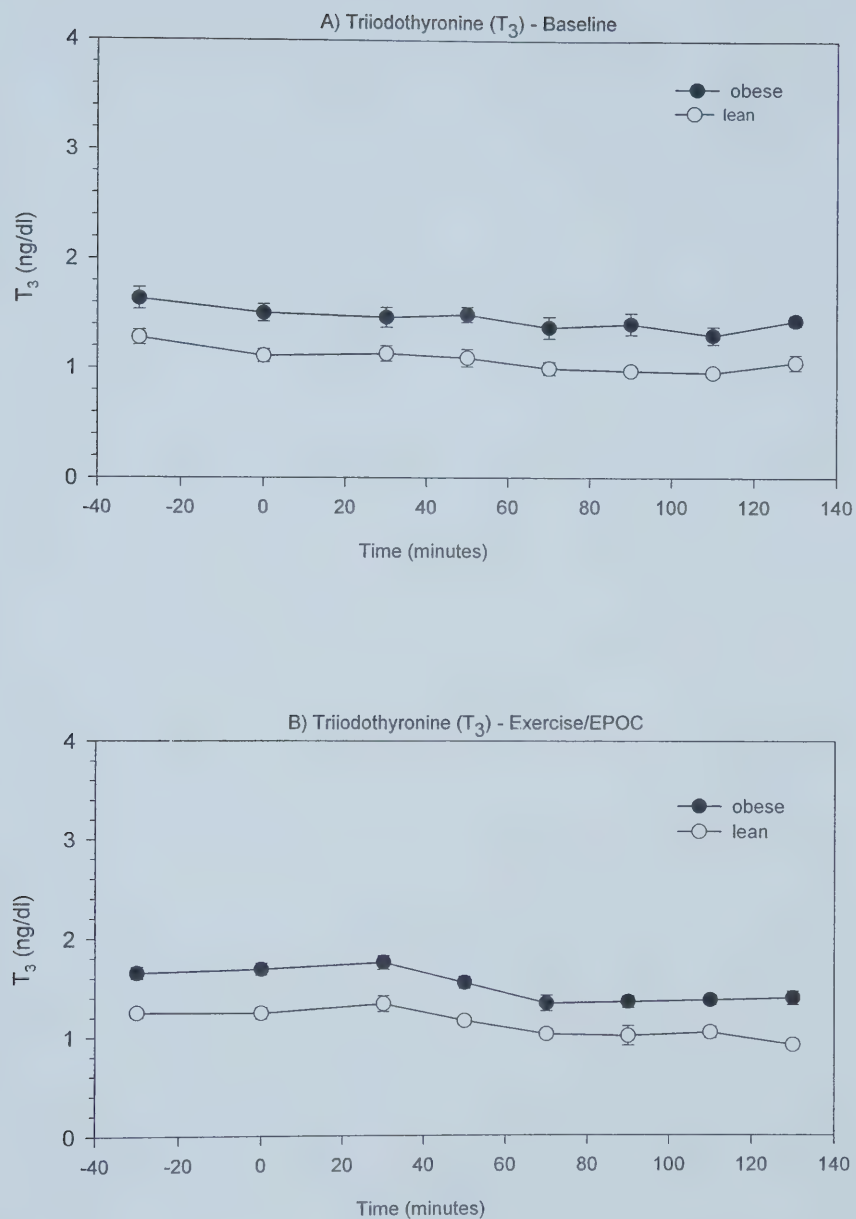


Figure 8. Serum T_3 levels during the Baseline (160 min rest) (A) and during Exercise/EPOC (30 min rest, 30 min exercise at VT, 100 min recovery) (B) conditions in 7 obese (black circle) and 6 lean (white circle) subjects. Values are expressed as mean $\pm$ SD

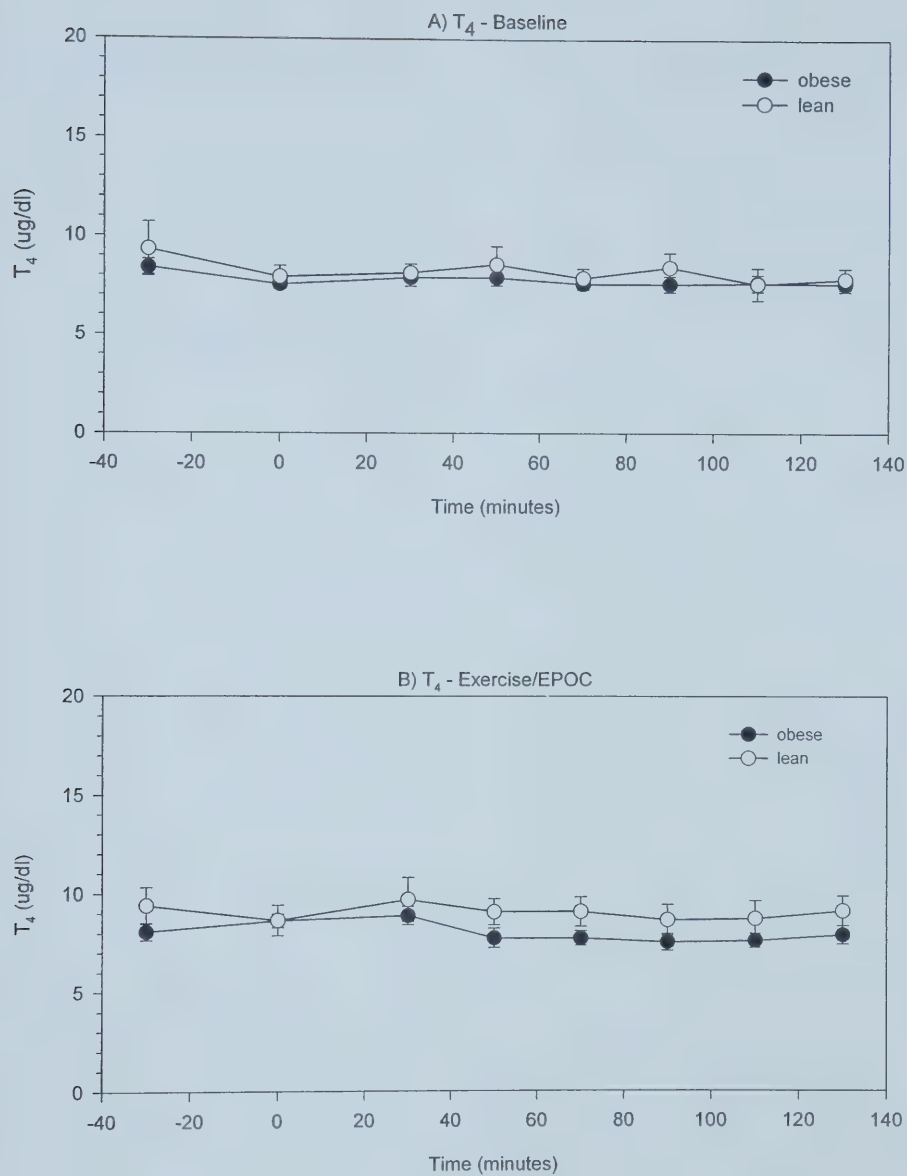
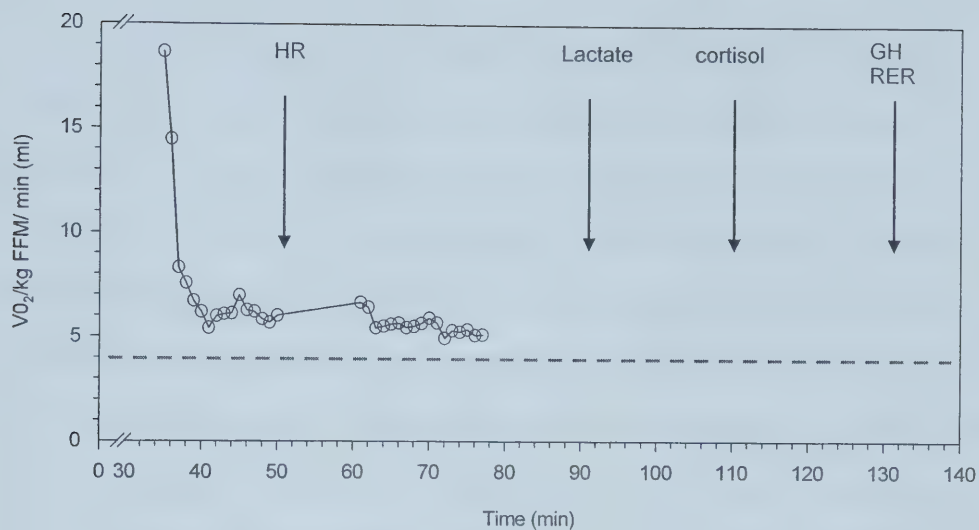


Figure 9. Serum T_4 levels during the Baseline (160 min rest) (A) and during Exercise/EPOC (30 min rest, 30 min exercise at VT, 100 min recovery) (B) conditions in 7 obese (black circle) and 6 lean (white circle) subjects. Values are expressed as mean $\pm$ SD.

A) Lean



B) Obese

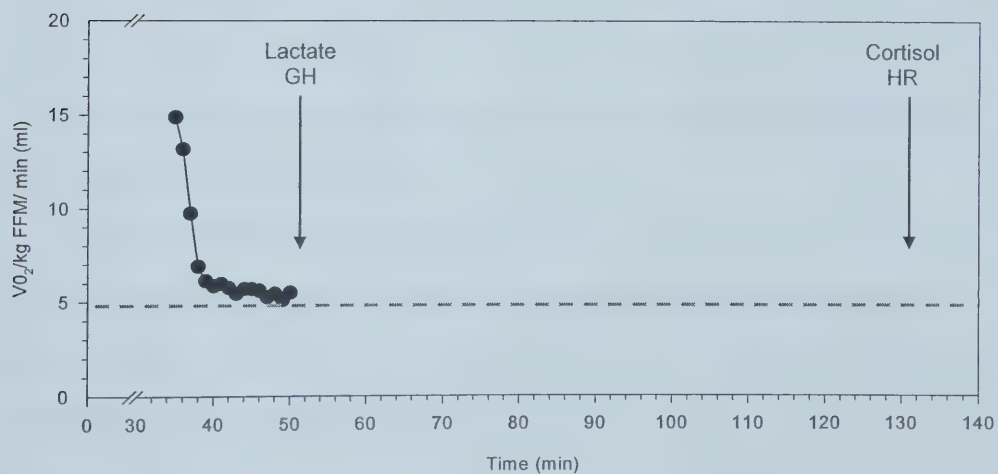


Figure 10. Recovery of HR, lactate, cortisol, GH, and RER in relation to the cessation of EPOC in lean (A) and obese (B) groups. Arrow indicates time at which differences between Baseline and Exercise/EPOC were no longer significant.

Chapter 5: Discussion

Responsiveness of the metabolic energy system to changing demands is differentially regulated in healthy weight versus overweight people such that a permissive metabolic environment may favour a propensity toward obesity. Two groups of subjects, obese and non-obese, exercised on a cycle ergometer for 30 minutes, standardized against their subject-specific anaerobic threshold (power output that elicited ventilatory threshold for carbon dioxide). This exercise prescription provided sufficient stimulus to stress aerobic metabolism and to elicit significant changes in the counter-regulatory hormones and EPOC response. The recovery period from exercise was the focus of this investigation. It may be during the recovery period rather than during exercise that differences in how the body copes with changing energy demands and prioritizes metabolic fuel selection become most apparent. Therefore, the purpose of this investigation was to examine the EPOC and metabolic hormone response during recovery from exercise in a cohort comparative analysis between groups of people distinguished by amount of body fat.

The results of this study suggest that differences in body composition lead to a different group response in EPOC and in the variables known to influence EPOC. Exercise induced a response in both lean and obese groups for lactate, RER, cortisol, and GH but did not elicit any changes in temperature, T_3 , or T_4 .

EPOC

This study showed that the magnitude of oxygen consumed post exercise was significantly different between lean and obese subjects when exercise intensity was set at similar ventilatory thresholds. Total magnitude of oxygen consumption in the lean group exceeded the obese group by 32.1 ml/kg FFM/ min ($p=0.007$). It is interesting to

note that in terms of absolute $\dot{V}O_2$, the obese subjects, owing to their larger body size (i.e. greater FFM), were significantly higher than the lean group during Baseline ($p<0.000$); but consumed significantly less oxygen during EPOC ($p=0.047$). This demonstrates either a blunted oxidative response from the obese or greater oxidative potential in the lean.

During the submaximal exercise bout, $\dot{V}O_2$ was similar between lean and obese groups (34 vs. 36.4 ml/kg FFM/min) suggesting that the higher EPOC values in the lean group reflect the recovery period rather than the exercise intensity. It is important to stipulate that both groups exercised at the same relative intensities ($\dot{V}E/\dot{V}CO_2$ threshold) and similar power outputs. In a higher intensity condition, there will be a higher starting point in the recovery period and $\dot{V}O_2$ will have further to fall to return to baseline, leading to a greater EPOC magnitude. As markers of stress, heart rate, lactate, and RER during exercise (Time30) were similar and not significantly different between groups during the exercise load.

In terms of EPOC response, neither group demonstrated a prolonged EPOC: the obese returned to baseline values by 28 minutes (Time58) and the lean group by 47 minutes (Time77) after the cessation of exercise. Duration of EPOC was longer in the lean ($p=0.008$) and probably accounted for the significant difference in magnitude.

Mechanisms

The mechanisms contributing to the rapid component of EPOC are temperature (18,43,44,51), lactate (43,44), and RER (10,24,111). Between session comparison of Baseline vs. Exercise/EPOC and between group comparison of lean vs. obese were conducted on each variable. Between session analysis was performed on each group separately to determine the duration that the variable remained elevated above resting

levels and between group analysis was performed to compare the obese to the lean response.

Temperature

Increased temperature has been suggested to partially account for EPOC by increasing cell respiration and decreasing phosphorylative coupling efficiency in the mitochondria (44). Gaesser and Brooks (44) attribute temperature as the most important factor affecting the slow component of EPOC (44) and a number of investigators have found elevated temperature following exercise (43,48,51). Other authors report a minor role for the effect of temperature on either the rapid or slow component of EPOC (67,87). In this study, exercise did not elicit significant changes in tympanic temperature compared to Baseline. As well, no significant differences were observed between lean and obese during Exercise/EPOC ($p=0.785$), including peak response ($p=0.329$). However, it is possible that measuring tympanic temperature was not sensitive enough to detect a change. Therefore, rectal or more specifically, muscle temperature (44) may more closely correlate with EPOC.

Lactate

The fate of lactate post-exercise (oxidation and conversion to glycogen) is energy requiring and occurs in the presence of oxygen. Lactate was originally thought to account for the majority of EPOC but contemporary views hold that lactate may not even correlate with EPOC (44). The temporal relationship between EPOC and lactate levels has presented contradictory evidence whereby lactate recovery precedes $\dot{V}O_2$ recovery as well as $\dot{V}O_2$ recovery preceding lactate recovery (44). In this study, the time-course of lactate recovery to non-significant values coincided with the cessation of EPOC (within a 20 minute sampling frequency) in both groups. In the lean, lactate recovered between

Time70-90 and the cessation of EPOC occurred at Time77. In the obese group, lactate recovered between Time50-70 and the cessation of EPOC occurred at Time58.

Blood lactate between lean and obese groups was not significantly different during the Exercise/EPOC session ($p=0.274$). Although not significantly different, a slightly higher peak response was observed in the lean (6.0 ± 2.9 mM) compared to the obese (4.6 ± 1.8 mM) group and may have a physiological impact that could slow recovery in the lean group.

Appearance of lactate in blood is a qualitative appraisal of glucose metabolism and is described in abnormalities of metabolism such as in obesity. Obesity, particularly with insulin-resistance, is associated with lactate overproduction in response to overnight fasting (69) and after oral glucose ingestion (93). In this study, there was no difference in lactate production between lean and obese subjects in response to exercise, and therefore, is in contrast to these findings. Other investigators have also found similar lactate concentrations in obese and lean subjects in response to different exercise protocols (3,103,116). In a study by Vettor et al. (116), lean and obese subjects exercised to volitional exhaustion and resulting lactate concentrations were similar (although slightly higher in the lean group: lean= 7.5 ± 0.6 mM and obese= 5.6 ± 0.7 mM). In addition, Lovejoy et al. (69) demonstrated a marked decrease in the capacity of obese subjects for acute lactate generation following an oral glucose load and Stunff et al. (104) reported increased gluconeogenesis from plasma lactate in obese compared to lean children. Thus, the trend towards lower lactate concentration in obese subjects may reflect a greater tendency toward lactate conversion to glucose (104).

Heart rate

Heart rate, although not indicated in the literature as a factor affecting EPOC, was measured as a response variable since elevated heart rate increases metabolism. The two groups did not differ from each other during Baseline ($p=0.956$) or Exercise/EPOC ($p=0.552$). However, between session analysis of the lean and obese comparing their Exercise/EPOC session against their Baseline sessions, revealed faster recovery rates for the lean group. The lean subjects recovered to resting levels by Time50, while the obese subjects remained elevated above resting levels until Time130. This finding is similar to the results reported by Astrup et al. (7) who studied obese women before and after weight normalization (postobese state) and found higher 24-hour heart rates when compared with matched controls. The authors concluded that postobese women have enhanced sympathetic nervous system (SNS) activity, which is responsible for the elevated heart rates observed (7). As well, in conditions associated with obesity, hypopituitarism and untreated GH deficiency, augmented or hyperactivity of the SNS in adults have been demonstrated by direct neural recordings (49,105).

RER

Lower RER during recovery compared to rest is associated with a post-exercise shift toward fat metabolism (24,111). Both groups demonstrated this trend (Figures 5a,b), although this was not significant in the obese and RER was at or above Baseline values after 65 minutes post-exercise (Time95). In the lean group, RER was below resting levels throughout recovery, which was significant at Time60-85 and Time96-125. This apparent difference in substrate oxidation in lean and obese during recovery is evident in a comparison between groups during Exercise/EPOC (Figure 5c). RER was lower in the lean compared to obese intermittently at Time60-80, Time115-120, and Time125-130. This discrepant response in RER further suggests a blunted oxidative

response in obesity, which supports the long held notion linking body fatness and muscle fibre-type profile to substrate oxidation (117), although many studies have failed to find significant correlations through measurement of RER (32,46,54).

The rapid component of EPOC, therefore, was associated with changes in lactate, heart rate, and RER, but not body temperature. The recovery of lactate possibly coincided with the cessation of EPOC (within 20 minutes) in both the lean and obese groups. Heart rate recovery occurred prior to the cessation of EPOC in the lean group but remained elevated until Time130 in the obese. Lower RER values were significant in the lean group only and persisted beyond the cessation of EPOC, indicating a more pronounced shift toward in fat oxidation in this group.

Hormones

The slow component of EPOC is attributed to elevated levels of metabolic hormones. Catecholamines (not measured), growth hormone (GH), cortisol, and thyroid hormones are stimulatory to lipolysis and would thus increase EPOC (18). During exercise, the magnitude of increase in GH and cortisol is related to exercise intensity and duration and slowly decline to resting levels after the cessation of exercise. As well, there may be an exercise threshold of intensity whereby these hormones increase significantly in the post exercise period and this may relate to the non-linear changes observed in EPOC (35,111).

The time course of recovery of these hormones in this study did not coincide with the cessation of EPOC. In the obese group, the cessation of EPOC occurred at 28 minutes post exercise (Time58) around the same time that GH returned to baseline levels (Time50-70 minutes) but cortisol remained elevated throughout (still significant at Time130). The end of EPOC occurred at 47 minutes post exercise (Time77) in the lean group, while GH remained elevated beyond the last sampling point (Time130) and

cortisol returned to resting levels at Time90-110. The difference in the recovery rates between lean and obese groups for GH and cortisol was related to significant group differences in the magnitude of these hormone levels: the obese group was statistically higher for cortisol and lower for GH.

Although thyroid hormones can change very quickly in response to an energy surplus or deficit, the thyroid axis is buffered against large fluctuations in energy status (32) and likewise, little variation was observed between Baseline and Exercise/EPOC conditions. Exercise did not elicit a significant increase in circulating levels of thyroid hormones compared to the Baseline session in the lean or obese groups. T_4 concentrations during Exercise/EPOC rose insignificantly from the average Baseline level of $7.8 \pm 0.3 \mu\text{g/dl}$ to a peak during exercise of $8.9 \pm 1.2 \mu\text{g/dl}$ in the obese group and from 8.2 ± 0.6 to $9.7 \pm 2.7 \mu\text{g/dl}$ in the lean group. Between group analysis revealed basal concentrations of T_3 in obese significantly higher than in lean but this difference was preserved during Exercise/EPOC and the difference (EPOC-Baseline) was minimal and non-significant. The higher resting level in the obese is suggested to reflect chronic overfeeding (1,94,97) but analysis of 3-day dietary intake revealed similar caloric intake in the obese compared to the lean group. Nonetheless, T_3 levels in the obese were within normal range and consistent with the results found in literature (1,97).

Overall, exercise induced a response in both lean and obese groups for lactate, RER, cortisol, and GH but did not elicit any changes in temperature, T_3 , or T_4 . The cessation of EPOC in the lean group possibly coincided with the recovery of blood lactate (within a 20 minute sampling frequency) and preceded the recovery of RER, cortisol, and GH to resting conditions. In the obese group, the recovery of lactate and GH levels possibly coincided with the end of EPOC, while cortisol remained elevated thereafter. Hence, the variables suggested to account for the EPOC response did not coincide with the time-course of EPOC and was different between groups. Although, in

the obese, lactate and GH recovery paralleled VO_2 recovery, and was, therefore, possibly underlying the EPOC response, this was not consistent in the lean subjects. The recovery of RER, heart rate, cortisol, and GH occurred at different time points in the obese and lean subjects, suggesting that the difference in body composition between groups attribute to a different impact of the variables influencing EPOC.

Obese vs. Lean

During the Baseline condition, there were no significant differences between lean and obese groups in cortisol, GH, and T_4 , but a significantly greater T_3 concentration in the obese. Although some studies have reported mild cortisol excess associated with obesity (15,35), the obese group in this study demonstrated normal resting (late afternoon) levels of $9.7 \pm 1.9 \mu\text{g/dl}$ (and statistically similar concentrations compared to the lean group at $5.8 \pm 1.7 \mu\text{g/dl}$). The obese were also within the normal range, 0.0-7.0 ng/ml, for GH, although the lean group demonstrated basal levels ($3.3 \pm 7.4 \text{ ng/ml}$) higher than the obese group ($0.7 \pm 0.5 \text{ ng/ml}$). This was not statistically significant but given that GH in the lean was four times the level in the obese, the physiological significance remains unclear. Resting levels of T_4 in the range of 6.1-11.8 $\mu\text{g/dl}$ are considered normal. Mean concentration for T_4 in the obese group was $7.78 \pm 0.7 \mu\text{g/dl}$ and the lean group was $8.2 \pm 0.6 \mu\text{g/dl}$. Mean T_3 concentrations in the obese group was 1.46 ng/ml compared to 1.08 ng/ml in the lean. T_3 levels in obese was significantly higher but within the normal range of 0.8-2.0 ng/dl.

The catecholamine response to exercise was not assessed due to the unavailability of the High Performance Liquid Chromatography (HPLC). From a review of literature, it appears that catecholamine concentrations consistently recover prior to the return of oxygen consumption to resting levels (18). Nonetheless catecholamines present an important contribution to glycolysis, gluconeogenesis, and lipolysis (18) and

Frey et al. (43) reported a moderate correlation between norepinephrine (NE) and EPOC ($r=0.78$).

Catecholamines are a marker of sympathetic activity and energy expenditure and the development of obesity can depend on decreased SNS activity. But a depressed SNS has not been convincingly demonstrated. In fact, many studies report augmented SNS activity (refer to the section on heart rate) associated with obesity. This occurs despite impaired sympathetic mediated fat utilization in response to β -adrenergic stimulation (16,96) demonstrated by pharmaceutical agents (96) or by exercise (50).

In response to the exercise stimulus, the obese group demonstrated blunted peak GH levels and higher peak cortisol levels compared to the lean group. Peak concentrations occurred at the end of exercise (Time30) and were 13.4 ± 12.2 ng/ml in the obese and 44.0 ± 22.2 ng/ml in the lean for GH and 25.5 ± 7.7 μ g/dl in the obese and 13.1 ± 5.3 μ g/dl in the lean for cortisol. This pattern persisted into the recovery period and significant differences still persisted 100 minutes post exercise (Time130). The GH response to exercise observed in this study agrees with the results presented by Jungmann et al. (61), Gustafson et al. (50), Garlaschi et al. (45), Weltman et al. (120), Vettor et al. (116), and Holt et al. (55), although only Jungmann et al. (61) report both blunted GH and higher cortisol response to exercise. Gustafson et al. (50) and Garlaschi et al. (45) also measured cortisol but found comparable or no change in cortisol levels. These studies, however, are difficult to compare since they investigate different subject groups. Jungmann et al. (61) compared less vs. more obese subjects based on levels of somatomedin C greater than or less than 0.7 U/ml; Garlaschi et al. (45) compared obese vs. diabetic vs. endocrinologically normal children; and Gustafson compared obese to normal women. As well, the exercise protocols in the latter 2 studies were 10 minute durations (at 70 and 50% $\dot{V}O_{2\max}$, respectively), possibly inadequate to elicit a cortisol response.

The physiological implications of the GH and cortisol profile found in this study cannot be directly assessed. Measurement of metabolic enzymes rates, particularly PDH and malonyl CoA, would be necessary to infer altered substrate utilisation in the obese subject. Decreased FFA utilisation during recovery from exercise, however, is indirectly supported by a smaller EPOC magnitude compared to controls and higher RER (although intermittently at Time60-80, Time115-120 and Time125-130 minutes). As well, measurement of plasma FFA and glucose levels would be necessary to estimate fuel mobilization. Suppression of GH release is suggested to occur when circulating levels of FFA are high (30,72), and obesity, in particular the visceral phenotype, is often associated with excessive FFA levels at inappropriate times when fatty acids are unlikely to be oxidized (78). However, the Baseline condition presented no significant difference in GH between groups. The further release of FFA in response to exercise could relate to the reduced GH response during the Exercise/EPOC condition. But this possibility is challenged by Jungmann et al (61), who reported blunted FFA stimulation in response to both exercise and GHRH administration and by Cordido et al. (30) who reported a smaller GH response to the administration of acipimox (causes a reduction of plasma FFA) plus GHRH. Another explanation is that there may be an increased re-esterification of FFA during recovery from exercise related to greater LPL expression in adipose tissue of obese subjects (59,91).

Although GH and cortisol share similar and redundant functions in fuel mobilization, both stimulate gluconeogenesis and are lipolytic, obesity is associated with low levels of GH and excess cortisol. The explanation for the disparate direction of these hormones may focus upon the adipocyte, whereby GH acts through the hormone-sensitive lipase to mobilize FFA's and cortisol, although lipolytic, is also capable of expressing LPL activity to exert the opposite effect of promoting lipid accumulation (15). Thus, during periods of substrate affluence (particularly with high circulating levels of FFA's), anabolic

pathways in lipid metabolism are up-regulated, promoting the release of cortisol and inhibiting GH secretion.

The low concentration of GH in obesity, therefore, is in part mitigated by the chronic elevation of FFA levels. Accordingly, modulation of GH activity has been achieved through manipulation of FFA levels. A decrease level of circulating FFA's with the administration of acipimox (a lipolysis inhibitor) and with diet-induced weight loss has resulted in the normalization of GH secretion to GHRH. As well, increased levels of FFA's with the administration of heparin and associated with overfeeding in rats has led to a diminished response of GH to GHRH.

Although GH secretion is regulated by FFA levels and would therefore be a consequence of visceral fat accumulation (i.e., an adaptive phenomenon) the low levels of GH found in obesity may also reflect an impairment of the axis activity (72). The pathogenesis of GH insufficiency, as reported by Maccario et al. (72), could originate centrally (CNS-related) and hypothetically include increased somatostatin release or impairment of GHRH tone or peripherally and involve metabolic alterations in insulin, FFA, leptin, and/or glucose.

In relation to cortisol, Maccario et al. (72) speculate that deficient GH, and thereby impaired fatty acid mobilization and/or oxidation, is conditional to the hypersecretion of cortisol to enhance the sensitivity of adipose tissue to low circulating levels of GH (72). This is in contrast to Dieguez et al. (34) and Wajchenberg et al. (118) who argue for a role of GC's in the neuroregulation of GH secretion. That is, chronic cortisol hypersecretion induces perturbations in the function of the somatotroph axis. This may occur directly at the pituitary and hypothalamic level (34) and affect the synthesis and secretion of GH. Although acute administration of GC to normal subjects induces a transient increase in plasma GH levels, chronic exposure to excessive cortisol secretion or administration is shown to blunt GH response to stimulation tests (34). The mechanism may occur through increased

hepatic gluconeogenesis and abnormal glucose metabolism, leading to alterations in insulin and finally to the GH axis (118).

Summary

In response to the same relative exercise challenge, obese subjects have reduced oxygen consumption during recovery from exercise compared to lean controls. This is associated with diminished caloric expenditure, which is likely in the form of fat oxidation. Indirect evidence is established by RER values higher in obese than lean during Exercise/EPOC that are significant intermittently at Time60-80, Time115-120 and Time125-130. As well, during recovery, RER was significantly lower than Baseline in the lean group only. This process may be mediated by the response of GH, which is greater in the lean subjects. An elevated cortisol response and elevated heart rates were noted in the obese subjects and are associated with greater sympathetic activation during exercise and driving recovery. These findings suggests that body composition influences the metabolic response to energetic challenges and provides evidence in support of the hypothesis put forth in this study indicating diminished oxidative activity post-exercise in the obese compared to lean groups. This was observed as a reduced EPOC response to exercise as well as depressed GH and exaggerated cortisol responses.

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Appendix 1

Individual subject data

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Table 1: Characteristics of lean and obese subjects**A) anthropometric**

	Age (years)	Height (cm)	Weight (kg)	Fat mass (kg)	FFM (kg)	Body fat (%)
Obese						
1	34	184	108.2	35.2	73	32.5
2	39	175	106.7	45.6	61.19	42.7
3	39	183	114.3	34.6	79.62	30.3
4	35	177	89	27.2	61.72	30.6
5	37	178.6	97.9	25.0	72.89	25.5
6	39	178	117.5	42.2	75.32	35.9
7	30	184	89.4	24.8	64.65	27.7
Lean						
1	35	175.2	60.5	9.7	50.82	16
2	36	157.3	55.6	7.6	47.97	13.7
3	37	186	74.5	11.2	63.26	15.1
4	36	172	68.2	11.1	57.09	16.3
5	33	183	65.5	10.6	54.91	16.2
6	30	177	73.5	11.0	62.5	15

B) Cardiorespiratory Fitness

	V_{O₂peak} (l/min)	V_{O₂} /kg (ml/kg/min)	V_{O₂} (ml/kg FFM/min)	% V_{O₂} at VT
Obese				
1	3.46	32	47.4	70
2	2.43	22.8	39.7	72
3	3.34	29.2	41.9	60
4	2.8	31.5	45.4	67
5	3.89	39.7	53.4	71
6	3.14	26.1	41.7	60
7	3.48	38	53.8	72
Lean				
1	2.65	43.8	52.1	70
2	2.53	45.4	52.7	68
3	3.3	44.3	52.2	78
4	2.81	44.8	49.2	75
5	2.43	37	44.3	60
6	2.61	35.4	41.8	57

FFM = fat-free mass

Table 2: Oxygen Consumption (AUC) during Baseline (gross magnitude)

	Absolute (l/min)	Relative (ml/kg/min)	Relative FFM (ml/kg FFM/min)
Obese			
1	24.956	230.65	341.86
2	24.196	226.77	395.42
3	25.725	225.07	323.1
4	20.242	227.44	327.97
5	22.546	230.3	309.32
6	24.008	204.32	318.75
7	21.604	241.66	334.17
Lean			
1	18.649	308.25	366.96
2	17.848	321.01	372.07
3	16.746	224.78	264.72
4	19.056	279.41	333.79
5	*	*	*
6	18.294	248.9	292.33

* Unavailable due to problems with metabolic cart

Table 3: Oxygen Consumption (AUC) during Exercise/EPOC gross magnitude)

	Absolute (l/min)	Relative (ml/kg/min)	Relative FFM (ml/kg FFM/min)	EPOC Duration (minutes)
Obese				
1	12.212	112.87	167.29	33
2	7.269	68.13	118.79	11
3	6.133	53.66	77.03	9
4	10.196	114.56	165.2	35
5	1.127	11.51	15.46	2
6	10.755	91.53	142.79	33
7	11.864	132.71	183.51	37
Lean				
1	10.990	181.65	216.25	36
2	12.442	223.78	259.37	43
3	9.400	126.17	148059	34
4	13.620	199.71	238.57	50
5	*	*	*	*
6	13.943	189.7	222.8	46

*Unavailable due to problems with metabolic cart

Note: Baseline $\dot{V}O_2$ is the area under the curve (AUC) from Time35-130

EPOC is the AUC from Time35 to the cessation of EPOC (defined on page 6)

Table 4: Three Day Energy Intake

	Protein (g)	Protein (%)	CHO (g)	CHO (%)	Fat (g)	Fat (%)	Alcohol (%)	Calories
Obese								
1	116.1	13.3	466.9	54.3	111.3	29	0	3396
2	76.3	13.3	313.2	51.3	96.3	35.3	0	2360
3	98.8	15	356.6	51	102.6	34	0	2701
4	*	*	*	*	*	*	*	*
5	73.6	16.7	217.1	52.7	57	30.7	0	1713
6	83.0	22	178.0	49	48.8	29	0	1462
7	59.9	12.3	246.6	54.3	69	33.3	0	1808
Lean								
1	99.1	17	374.6	63	53.3	20	0	2297
2	*	*	*	*	*	*	*	*
3	99.18	14	329.9	47.3	100.8	33	0	2723
4	90.5	15.3	338.6	58	49.8	18.7	8	2304
5	117.6	15	306.7	41.3	122.3	36.7	7	2994
6	141.5	17.3	323.5	39	140.7	38.7	4	3239

* Subjects did not return dietary intake record

Table 5: Heartrate response during Baseline and Exercise/EPOC**A) Baseline**

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	75	80	75	69	71	77	67	66
2	80	70	70	72	65	67	69	67
3	62	59	63	63	60	61	64	64
4	66	61	63	64	65	55	63	62
5	77	71	79	64	58	58	61	56
6	63	61	60	60	55	53	50	57
7	66	58	57	54	54	51	58	56
Lean								
1	62	65	50	47	44	57	47	47
2	68	75	70	71	62	63	75	67
3	68	69	64	64	60	70	63	61
4	70	70	59	65	62	60	61	63
5		82	71	86	66	73	65	65
6	80	71	76	63	65	65	65	65

B) Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	83	110	137	94	73	89	74	73
2	84	112	163	103	87	89	82	82
3	67	118	167	92	78	71	68	69
4	69	124	170	85	85	75	78	66
5	78	130	143	108	79	72	72	73
6	67	105	149	102	76	73	73	67
7	69	85	169	78	67	62	64	64
Lean								
1	57	112	143	80	52	57	55	53
2	65	97	160	108	86	95	100	97
3	66	111	185	102	76	67	71	73
4	66	107	158	72	62	64	68	70
5	96	131	150	75	75	85	68	77
6	73	125	129	87	79	67	64	81

Table 6: Temperature during Baseline and Exercise/EPOC**A) Baseline**

	Time -30	Time 0	Time 30	Time 50	Time 90	Time 120	Time 130
Obese							
1	36.7	36.5	36.8	37	36.5	36.3	36.6
2	36.9	36.6	36.2	36.1	35.3	35.4	37.2
3	37.4	36.9	36.7	36.4	36.8	36.8	36.8
4	36.2	36.5	36.5	-	-	-	-
5	36.5	36.6	36.3	36.3	36.4	36.6	-
6	35.3	34.5	35	34.2	-	-	34.8
7	36.6	36.8	36.8	36.9	37	36.5	36.4
Lean							
1	36.8	35.4	36.6	36	36.2	36.2	36.2
2	36.6	36.6	36.4	36.3	*	*	35.7
3	36.2	35.9	35.5	35.7	36.7	36.5	36.5
4	36.3	36.4	35.8	36.1	36.4	36.7	36.9
6	36.6	-	36.6	-	-	-	36.6

b) Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 90	Time 120	Time 130
Obese							
1	36.7	36.5	35.6	36.7	37	36.9	37
2	36.6	36.4	37.1	36.3	36.4	36.3	36.7
3	37.2	36.5	36.3	36.1	36.9	36.1	36.1
4	36.5	36.5	36.5	-	36.2	36.6	36.3
5	36.7	36.7	37.1	37.2	36.8	36.5	-
6	35.5	36.1	35.5	35	35.3	35.5	34.5
7	37.3	37.1	36.9	36.6	37.3	37	37
Lean							
1	36.6	36	37.2	35.8	36.7	36.9	35.9
2	36.6	36.7	38.3	35.6	36.3	36.5	35.6
3	36.3	36.5	37.6	36.8	35.8	36	36.5
4	36.1	35.6	35.1	35.6	36.4	36.2	36
6	36.4	36	36.3	36.5	36.3	36.3	36.2

Table 7: Lactate response during Baseline and Exercise/EPOC**A) Baseline**

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	0.604	0.657	0.242	0.189	0.175	0.309	0.282	0.162
2	0.606	0.481	0.481	0.435	0.621	0.45	0.59	0.606
3	0.142	0.174	0.254	0.206	0.238	0.222	0.318	0.286
4	0.995	0.731	1.058	0.855	0.84	0.98	1.026	1.151
5	0.0784	0.7824	0.0784	0.0304	*	*	*	*
6	0.292	0.444	0.237	0.278	0.375	0.402	0.472	0.154
7	0.376	0.282	0.095	0.296	0.149	0.202	0.162	0.242
Lean								
1	0.317	0.204	1.576	0.373	0.199	0.175	0.345	0.345*
2	0.552	0.294	0.249	0.34	0.219	0.385	0.476	0.34
3	0.216	0.173	0.188	0.315	0.415	0.216	0.315	1.068
4	0.568	0.1	0.734	0.082	0.036	0.188	0.112	0.234
5	1.338	0.574	0.588	0.178	0.178	0.023	0.518	0.079
6	0.475	0.984	0.348	0.306	0.532	0.461	0.419	0.291

* less than standard

B) Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	0.47	0.443	1.822	0.497	0.256	0.175	0.59	0.403
2	0.653	1.322	3.114	1.276	1.042	0.731	0.684	0.512
3	0.062	2.206	7.278	2.446	1.566	1.23	0.574	0.606
4	0.497	0.668	5.808	2.366	1.12	0.995	1.042	0.902
5	0.366	0.766	5.406	2.222	0.894	0.478	0.318	0.27
6	0.555	2.436	4.414	1.55	0.9	0.555	0.458	0.582
7	0.229	0.885	4.017	0.697	0.564	0.082	0.122	0.376
Lean								
1	0.897	1.279	5.055*	1.236	0.529	0.925	0.43	0.345*
2	0.583	4.528	10.568	5.287	3.13	2.024	1.402	0.962
3	0.898	1.807	6.935	2.304	1.224	0.898	0.614	0.685
4	0.112	1.417	3.13	0.719	0.932	0.674	0.583	0.34
5	0.136	0.829	2.908	0.532	0.207	0.263	0.136	0.277
6	0.334	3.446	7.689	2.767	1.847	1.055	0.786	0.914

Table 8: Hormones response during Baseline and Exercise/EPOC**A) Cortisol Baseline**

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	10.639	8.905	7.599	6.47	6.265	10.329	9.128	9.571
2	6.042	7.633	6.913	6.342	5.4	4.568	3.785	4.155
3	17.545	17.723	18.728	19.096	14.794	11.158	9.01	7.819
4	5.078	4.383	4.563	4.265	7.503	7.132	5.072	3.975
5	6.741	6.586	7.518	7.569	6.148	5.443	3.932	4.135
6	5.373	25.005	26.863	24.122	19.459	17.575	15.154	15.994
7	14.619	13.688	11.465	9.902	8.02	6.572	5.082	5.859
Lean								
1	7.577	8.416	7.254	8.092	6.743	6.403	6.105	5.404
2	10.664	9.069	9.022	8.118	5.78	5.045	4.09	5.039
3	10.511	8.894	7.185	5.613	4.88	3.683	3.085	3.807
4	6.844	6.745	5.652	4.601	3.931	3.333	2.396	2.266
5	4.968	8.062	6.123	4.892	3.898	2.685	2.347	2.49
6	7.839	5.955	4.555	5.757	6.199	4.407	5.339	5.121

B) Cortisol Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	10.687	7.553	10.312	16.339	12.335	9.429	9.13	10.533
2	7.101	6.106	22.001	19.219	11.673	8.637	8.383	7.42
3	13.877	16.031	25.631	23.235	28.455	26.353	20.107	18.592
4	6.719	5.282	29.005	35.826	23.712	16.465	16.751	14.132
5	14.249	9.794	31.177	34.926	24.487	23.151	20.4	14.563
6	9.871	10.25	26.334	27.144	24.014	18.236	13.615	13.621
7	7.138	18.288	33.899	26.431	17.849	14.158	11.673	10.87
Lean								
1	8.769	7.556	10.469	10.101	8.687	6.899	6.655	6.146
2	7.357	8.688	20.006	22.199	22.084	19.535	15.147	12.442
3	12.394	9.184	19.475	20.139	11.933	11.166	9.733	9.143
4	6.491	5.345	8.923	7.767	6.491	5.69	4.898	4.565
5	5.261	4.91	8.398	8.929	8.594	7.22	6.789	6.232
6	8.084	*	11.181	10.303	7.53	5.412	4.852	3.545

C) Growth Hormone Baseline

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	0.291	4.339	1.626	0.742	0.279	0.325	0.623	1.952
2	0.378	0.161	0.073	0.03	0.037	0.025	0.021	0.097
3	0.037	0.257	1.311	0.62	0.215	0.11	0.052	0.027
4	0.092	0.024	0.011	0.045	0.15	0.062	0.058	0.031
5	0.344	0.072	0.051	0.067	0.062	0.044	0.042	0.202
6	0.301	8.37	3.506	3.607	2.081	1.425	2.492	2.827
7	0.026	0.121	0.104	0.536	0.347	0.172	0.064	0.037
Lean								
1	8.108	28.38	15.2	10.05	5.408	3.063	1.812	1.083
2	0.184	0.215	0.327	1.343	1.777	0.799	0.309	0.241
3	0.059	1.496	2.356	1.31	0.923	0.322	0.173	0.138
4	0.217	0.334	0.203	0.149	0.098	0.092	0.112	0.079
5	32.02	27.98	8.513	3.46	1.987	0.834	0.531	0.279
6	0.266	0.172	0.089	0.127	0.119	0.088	0.084	0.147

D. Growth Hormone Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	0.282	4.967	7.939	2.812	1.079	0.493	0.224	0.155
2	0.06	0.074	4.89	1.759	0.504	0.195	0.099	0.055
3	0.008	0.013	9.263	4.01	2.211	0.86	0.239	0.166
4	0.21	0.059	3.728	1.761	0.428	0.19	0.099	0.055
5	2.337	0.46	27.095	8.836	3.373	1.619	0.583	0.276
6	0.036	0.481	6.112	3.441	1.85	0.856	0.671	0.791
7	0.143	2.266	34.459	10.087	3.831	1.591	0.792	0.31
Lean								
1	0.346	14.483	32.834	13.25	6.741	2.929	1.747	1.022
2	0.102	1.193	73.539	37.804	17.835	9.901	4.73	2.778
3	17.253	27.825	28.697	11.169	5.274	2.619	1.614	0.905
4	0.195	3.342	14.453	3.474	2.259	1.08	0.453	0.237
5	6.481	16.248	53.005	20.791	8.88	3.972	2.187	1.258
6	18.932	*	60.95	25.217	10.194	4.687	2.497	1.436

E) T3 Baseline

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	1.96	1.592	1.66	1.756	1.461	1.907	1.518	1.629
2	1.757	1.741	1.639	1.575	1.688	1.545	1.544	1.437
3	1.889	1.505	1.433	1.688	1.541	1.448	1.441	1.538
4	1.632	1.614	1.649	1.503	1.539	1.372	1.271	1.522
5	1.433	1.564	1.383	1.379	1.341	1.134	1.075	1.237
6	1.556	1.423	1.489	1.269	1.152	1.305	1.253	1.483
7	1.228	1.108	1.018	1.291	0.913	1.174	1.051	1.266
Lean								
1	1.047	1.018	0.807	0.73	0.808	0.881	0.869	0.861
2	1.585	1.383	1.264	1.294	0.903	0.992	1.094	1.082
3	1.271	0.97	1.277	1.1	1.201	1.095	0.983	1.39
4	1.155	1.152	1.2	1.131	0.992	0.919	1.026	1.026
5	1.332	1.09	1.175	1.15	1.036	1.025	0.936	1.033
6	1.278	1.064	1.086	1.177	1.066	0.964	0.876	0.958

F) T3 Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	1.782	1.713	2.031	1.72	1.483	1.544	1.46	1.684
2	1.735	1.862	1.643	1.681	1.388	1.353	1.481	1.224
3	1.935	1.797	1.833	1.535	1.552	1.579	1.53	1.566
4	1.567	1.686	1.78	1.646	1.274	1.181	1.365	1.412
5	1.591	1.535	1.483	1.43	1.244	1.294	1.317	1.181
6	1.513	1.787	1.836	1.589	1.512	1.384	1.323	1.447
7	1.474	1.459	1.677	1.272	0.938	1.155	1.144	1.244
Lean								
1	1.259	1.197	1.127	1.13	0.853	0.74	0.94	0.953
2	1.443	1.246	1.626	1.272	1.205	1.501	1.274	0.718
3	1.293	1.475	1.416	1.197	1.096	0.988	1.103	1.032
4	1.287	1.233	1.193	1.306	1.089	0.97	1.065	0.956
5	1.106	1.081	1.219	0.972	0.922	0.9	0.874	0.855
6	1.122	*	1.446	1.118	1.009	0.967	1.015	0.999

G) T4 Baseline

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	10.036	8.642	9.36	9.595	8.434	9.487	9.613	8.808
2	7.799	7.417	6.794	6.525	7.32	6.707	7.421	7.006
3	8.817	7.65	8.303	8.184	7.901	7.704	7.614	7.052
4	9.354	7.567	9.054	8.965	8.641	8.078	8.035	8.797
5	6.875	6.741	6.127	6.728	6.53	6.095	5.9	6.436
6	8.184	7.95	7.779	7.87	7.641	7.837	8.037	8.284
7	7.838	6.823	7.905	7.541	6.736	7.266	6.901	6.862
Lean								
1	7.527	7.815	8.045	6.933	7.461	8.84	6.791	6.756
2	16.008	9.215	10.006	11.065	9.198	11.503	9.824	8.603
3	8.285	7.243	7.404	8.19	7.429	7.387	6.674	8.714
4	7.015	6.665	6.875	6.515	6.632	6.913	6.225	6.528
5	7.565	6.709	8.048	7.135	6.997	6.707	5.646	6.87
6	9.626	9.956	8.523	11.72	9.65	9.377	10.478	9.692

H. T4 Exercise/EPOC

	Time -30	Time 0	Time 30	Time 50	Time 70	Time 90	Time 110	Time 130
Obese								
1	10.314	10.073	11.009	9.541	9.635	9.881	9.406	10.241
2	7.708	8.129	7.6	7.363	7.717	6.62	7.169	6.935
3	7.85	8.268	8.928	5.435	7.29	7.626	7.314	7.591
4	8.551	9.613	9.787	9.082	8.454	7.957	7.902	8.89
5	8.032	8.018	7.466	7.333	6.832	6.67	6.827	6.897
6	6.46	7.591	8.377	7.694	6.866	7.092	8.135	7.598
7	7.719	8.858	9.093	7.766	7.339	6.926	6.566	7.16
Lean								
1	7.595	7.929	8.007	7.788	7.672	7.115	6.621	6.889
2	13.07	11.553	11.898	10.337	10.736	11.15	10.131	11.92
3	10.41	8.925	9.277	10.251	10.18	8.82	8.449	8.79
4	8.085	7.652	7.592	8.199	8.281	7.677	8.412	10.16
5	7.119	7.239	7.344	6.983	6.591	6.597	6.527	7.481
6	10.212	*	14.106	10.861	11.015	10.616	12.227	9.414

* technical difficulty during blood draw, therefore, unavailable.

Table 9: RER (averaged 5 minute intervals) during Baseline and Exercise/EPOC

A) Baseline

Time (min)	35	40	45	50	55	60	65	70	75	80	85	90	95	100	105	110	115	120	125
Obese																			
1	0.8	0.78	0.84	0.83	0.83	0.83	0.83	0.83	*	0.82	0.8	0.81	0.79	0.79	0.78	0.76	0.76	0.79	0.76
2	0.8	0.792	0.792	0.796	0.796	0.796	0.826	0.826	0.804	0.768	0.634	0.65	0.642	0.642	0.65	0.712	0.696	0.694	0.696
3	0.78	0.81	0.79	0.77	0.79	0.79	0.83	0.83	0.81	0.78	0.79	0.76	0.75	0.75	0.79	0.82	0.79	0.73	0.73
4	0.832	0.868	0.886	0.888	0.888	0.796	0.966	0.966	0.928	0.818	0.97	0.906	0.912	0.912	0.966	0.932	0.838	0.652	0.644
5	0.826	0.828	0.822	0.824	0.85	0.85	0.806	0.806	0.77	0.822	0.73	0.672	0.644	0.644	0.708	0.804	0.844	0.786	0.752
6	0.722	0.728	0.74	0.758	0.754	0.754	0.76	0.76	0.78	0.748	0.786	0.76	0.764	0.764	0.778	0.784	0.786	0.736	0.722
7	0.738	0.81	0.726	0.768	0.754	0.754	0.78	0.78	0.772	0.752	0.81	0.806	0.806	0.806	0.836	0.872	0.804	0.762	0.832
Lean																			
1	0.778	0.858	0.808	0.844	0.818	0.818	0.912	0.912	0.748	0.71	0.686	0.698	0.688	0.688	0.734	0.774	0.796	0.806	0.742
2	0.784	0.796	0.79	0.782	0.812	0.812	0.78	0.78	0.804	0.798	0.796	0.798	0.812	0.812	0.786	0.852	0.834	0.758	0.792
3	0.792	0.748	0.746	0.82	0.834	0.834	0.802	0.802	0.808	0.842	0.784	0.816	0.806	0.806	0.804	0.82	0.838	0.892	0.89
4	0.814	0.822	0.808	0.824	0.828	0.828	0.814	0.814	0.798	0.776	0.814	0.828	0.77	0.77	0.768	0.75	0.766	0.742	0.788
5	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
6	0.856	0.788	0.886	0.744	0.756	0.756	0.86	0.86	0.892	0.868	0.908	0.894	0.874	0.874	0.902	0.894	0.884	0.88	0.928

* unavailable due to problems with the metabolic cart during testing

B) Exercise/EPOC

Time (min)	35	40	45	50	65	70	75	80	85	95	100	105	110	115	120	125	130	
Obese																		
1	0.784	0.988	1.095	0.978	-	0.758	0.7	0.73	0.733	-	0.782	0.792	0.776	0.788	-	-	0.788	
2	0.914	0.866	0.848	0.798	0.724	0.706	0.738	0.738	0.618	0.596	0.596	0.544	0.538	0.535	-	-	0.78	
3	0.918	0.826	0.824	0.814	-	0.823	0.82	0.778	0.812	0.908	0.84	-	0.875	0.892	0.858	0.834	0.842	
4	1.02	0.872	0.794	0.744	0.748	0.778	0.824	0.824	0.78	0.81	0.868	0.928	1.014	0.958	0.912	0.893	0.964	
5	0.686	0.632	0.706	0.686	0.752	0.764	0.778	0.782	0.756	0.752	0.764	0.778	0.782	0.756	0.764	0.816	0.77	
6	0.862	0.796	0.75	0.72	0.734	0.722	0.73	0.732	0.732	0.796	0.782	0.784	0.772	0.788	0.782	-	0.796	
7	0.918	0.804	0.784	0.788	0.708	0.706	0.734	0.692	0.642	0.83	0.85	0.802	0.786	0.832	0.878	0.953	0.93	
Lean																		
1	0.996	1.042	0.76	0.984	0.578	0.546	0.578	0.618	0.64	0.766	0.726	0.73	0.758	0.748	0.762	-	0.756	
2	0.91	0.984	0.822	0.666	0.678	0.646	0.69	0.706	0.726	0.753	0.66	0.682	0.68	0.678	0.672	0.71	0.76	
3	0.81	0.714	0.666	0.668	0.644	0.632	0.638	0.652	0.684	0.728	0.69	0.734	0.736	0.76	0.762	0.772	0.76	
4	0.898	0.798	0.73	0.766	-	0.765	0.686	0.684	0.65	0.76	0.71	0.658	0.632	0.618	0.626	-	-	
5	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*	*
6	0.97	0.866	0.85	0.824	0.688	0.67	0.674	0.652	0.632	0.78	0.714	0.706	0.718	0.718	0.726	0.75	0.74	

- unavailable due to a delay in calibration of metabolic cart

* unavailable due to problems with the metabolic cart during testing

Appendix 2: Calculations

1. Sample Size Calculation:

$$n = \frac{2 \times 7.9 \times (CV)^2}{(x)^2}$$

where: x = % difference between the means for 2 groups

CV = coefficient of variation, which is the standard deviation expressed as a percentage of the mean

Expected values for x and CV are EPOC results based on data from Frey GB and Mazzeo R, 1993 (43).

$$n = \frac{2 \times 7.9 \times (0.65)^2}{(0.7)^2}$$

$$n = 13.6$$

2. Decay of $\dot{V}O_2$ (half time) for the rapid component of the EPOC curve

Formula for exponential decay:

$$Y = Y_0 + ae^{-kt}$$

Where: $Y = \dot{V}O_2$ after the onset of recovery
 $Y_0 =$ resting $\dot{V}O_2$
 $k =$ rate constant
 $t =$ time

Lean

$$Y = 5.11 + 13.56e^{-0.57t}$$

$$T_{1/2} = 1.22 \text{ minutes (73.2 sec)}$$

Rapid component = 5 minutes

Slow component (linear regression line): $Y = -0.022x + 6.05$

Obese

$$Y = 4.62 + 10.26e^{-0.38t}$$

$$T_{1/2} = 1.82 \text{ minutes (109 sec)}$$

Rapid component = 5 minutes

Slow component (linear regression line): $Y = -0.049x + 6.01$

Note: the rapid component was determined by first determining the linear regression line of the slow component. This line was extrapolated to the y-axis. The point of intercept is the first data point of slow component. This was then confirmed by setting the equation of the fast component equal to the equation of the slow component and solving for t , which yields the intercept point.

Example: $5.11 + 13.56 e^{-0.57t} = -0.22t + 6.05$
 $t =$ intercept

Appendix 3

Data Collection Forms

PARTICIPANT INFORMATION LETTER

Postexercise oxygen consumption and metabolic hormone profile in lean and obese men.

Investigators:	Tina Wong 492 – 8739 E455 Van Vliet Centre kwong@ualberta.ca	Vicki Harber, Ph.D. 492 – 1023 E475 Van Vliet Centre vharber@per.ualberta.ca	Gordon Bell, Ph.D. 492 - 2018 E473 Van Vliet Centre gbell@per.ualberta.ca
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Why are we doing this study?

We are examining the recovery period from exercise in a comparison between two groups of men that differ by amount of body fat. The purpose of this study is to investigate the relationship between level of body fat and energy expenditure. The information obtained from this study will be used to examine why some people are susceptible to weight gain and resistant to weight loss. This study is being performed for thesis research.

Background

An efficient metabolism in the form of low energy expenditure has been described in the tendency toward obesity. Recovery from exercise is a component of total energy expenditure and, therefore, "excess postexercise oxygen consumption" (EPOC) and the hormones regulating energy expenditure will be assessed. The measurements we will be taking are oxygen consumption, temperature, urine and blood samples for hormone and lactate levels. These factors will be analyzed to relate the mechanisms of energy expenditure to body composition (%body fat level).

Procedures

You will first complete a PAR-Q (Physical Activity Readiness Questionnaire) to ensure you are a suitable candidate for maximal exercise testing ($\text{VO}_{2\text{max}}$). We will also measure body mass index (a measure of the ratio of weight to height) to get an estimate of whether or not you are within the weight groups of interest for this study. If you are eligible to continue, for your first session (2.5 h), you will undergo hydrostatic (underwater) weighing to determine body fat level and $\text{VO}_{2\text{max}}$ testing to determine fitness level. These measures will be used to determine your %body fat and fitness level. For those subjects that remain eligible, a 3-day diet record will be handed out and you will be provided with instructions on accurate record keeping. When you return the diet record, we will make an appointment for your next visit, which will have you come into the lab in the afternoon for a measurement of resting (**Baseline**) metabolism (3 hours). For the final session, you will come in for a 30 minute exercise and recovery session (**Exercise/EPOC**) (3 hours). See the diagram provided for an overview of the study.

1. BMI

This is the ratio of a person's weight to height (kg/m^2) and therefore involves measurement of weight using a standard physician's weight scale and standing height. BMI will be used only in initial recruitment to quickly screen and exclude people who are clearly outside the weight ranges of interest.

2. Maximal oxygen consumption ($\text{VO}_{2\text{max}}$)

Maximal oxygen uptake ($\text{VO}_{2\text{max}}$) will be measured during a progressive, incremental bike test to exhaustion (until the person says that he cannot continue). The exercise intensity is quite light at the beginning of the test, and becomes more difficult every minute or two. The actual test usually lasts for about 12-15 minutes, with an additional 5-10 minutes of warm-up and cool-down exercise before and afterwards. During the test, expired gases will be collected using a special breathing apparatus (mouthpiece). Heart rate will be monitored continuously with a heart rate monitor (a belt that fits around your chest). This test will be performed on a stationary bike and you will be asked to start cycling at 60 rpm at a resistance of 0.5 kp. The resistance will increase by 0.5 kp every 2 minutes. Near the end of the test the resistance will be increased every minute until you are exhausted and/or stop the test. (Time=1 hour).

3. Hydrostatic(underwater) weighing

This is one of most accurate methods for determining a person's percentage of body fat. This involves submersion under water so you will need to bring along a swimsuit or shorts (a changing facility is provided). Prior to entering the water tank, body weight will be measured on a balance-beam scale, height recorded, and lung volume measured by breathing into an apparatus. The underwater procedure involves being seated in a chair and lowered until complete submersion under water for approximately 10 seconds. You will be required to remain as still as possible and to not exhale the air from your lungs during this time. Upon being raised out of the water, you will be handed a breathing hose to exhale into. This is necessary to account for the air in the lungs, so that it is not added into the calculation of body fat. You will be placed under water 8-12 times. (Time = 1.5hours).

4. Assessment of dietary intake

You will be asked to keep a diary of all foods and liquids that you consume for a period of 3 days. This is 3 days in a row and will include a Saturday or Sunday. You will be given instructions and a sample diet record. You will need to be as explicit as possible. The detail required includes serving size, amount, brand name, and methods of cooking. This will be used to calculate total calories as well as the proportion of calories derived from carbohydrates, protein, and fats. As well, 1 day of the diet record will be photocopied for you and you will be asked to follow it for your meal plan on the day of the **Baseline** and **Exercise/EPOC** sessions.

5. Baseline Testing (**Baseline**)

This session will measure resting oxygen consumption, temperature, and blood hormone levels across a 3 hour period. This involves resting in a seated position for the first 80 minutes and then lying on your back for the next 100 minutes. An indwelling catheter will be inserted by a nurse practitioner prior to the start of the session. Blood samples will be collected at the times identified on the diagram. At each time point, 3 ml (less than 1 teaspoon) of blood will be collected. The total amount of blood collected from each subject will be 27 ml (approximately 2 tablespoons) for this session. Temperature will also be measured at the same time points by an ear thermometer. A canopy, that covers your head region, will be used to connect you to a metabolic cart that will measure oxygen consumption continuously. (Time = 3 h).

6. Exercise and Recovery testing (**Exercise/EPOC**)

This session will measure oxygen consumption, temperature and blood hormone levels across a 3 hour period. You will be asked to exercise on a stationary bike for 30 minutes at a moderately intense level. An indwelling catheter will be inserted by a nurse practitioner prior to the start of the session. Blood samples will be collected at the times identified on the diagram provided. At each time point, 3 ml (less than 1 teaspoon) of blood will be collected. The total amount of blood collected from each subject will be 27 ml (approximately 2 tablespoons) for this session. Temperature will also be measured at the same time points by an ear thermometer. During the exercise session and a portion of the recovery period, a mouthpiece will be used to connect you to the metabolic cart. For the remainder of the recovery period, a canopy, that covers your head region, will be used to connect you to a metabolic cart that will measure oxygen consumption continuously. As well, your heart rate will be monitored continuously with a heart rate monitor (a belt that fits around your chest. As depicted by the diagram, the protocol begins with a 25 minute rest period (-30 min until time -5 min), after which you will warm-up for 5 minutes (time -5 until time 0), exercise for 30 minutes at a preset intensity on a stationary bike (time 0 until time 30), and then rest (time 30 min until 150 min). Of the recovery period, the first 20 minutes you will sit in a chair and the remainder of the time you will take a lying position on a cot. (Time = 3 h).

Benefits

The benefits for participating in this study include a body composition assessment (hydrostatic weighing to determine % body fat level), a fitness assessment (exercise test to determine VO_2max), assessment of dietary intake, and general advice on exercise prescription. The testing involved in this study as provided by the University of Alberta would otherwise cost \$200.

Potential Risk

The procedures which present health risk are the maximal exercise test and blood collection via indwelling catheter. The VO_2max test involves maximal physical effort and as such it is possible, during and after the test, to experience symptoms such as abnormal blood pressure, fainting, lightheadedness, muscle cramps or strain, nausea, and in very rare cases (0.5 per 10,000 in testing facilities such as exercise laboratories, hospitals and physician's offices), heart rhythm disturbances or heart attack. While serious risk to healthy participants is highly unlikely, they must be acknowledged, and participants willingly assume the risks associated with very hard exercise. The exercise test will be administered by qualified personnel who are under the supervision of Drs. Vicki Harber and Gordon Bell. Personnel are trained to handle identifiable risks and emergencies, and have certification in CPR. Certifications can be produced if requested.

The blood collection procedure using an indwelling catheter will be performed by a registered nurse practitioner. There is a small risk of infection, bruising, and hematoma (bleeding) at the site if not properly cared for. Standard laboratory safety procedures are in place and will be adhered to.

Confidentiality:

To ensure confidentiality, personal information will be coded and stored in a locked file cabinet to which only the investigators have access. Normally, information is retained for a period of five years post publication, after which it will be destroyed.

FOIPP:

The University of Alberta creates and collects information for the purposes of research and other activities directly related to its educational and research programs. All participants in research projects are advised that the information they provide, and any other information gathered for research projects, will be protected and used in compliance with Alberta's Freedom of Information and Protection of Privacy Act.

Third Party Contact:

If you wish to speak with someone who is not involved with this study, please call Dr. Debra Shogan, Associate Dean (Research and Graduate Studies), Faculty of Physical Education and Recreation, at 492-5910.

Voluntary Participation:

You are free to withdraw from the study at any time. If you decline to continue or you withdraw from the study your information will be removed from the study upon your request.

CONSENT FORM

Part 1 (to be completed by the Principal Investigator):

Title of Project: Postexercise oxygen consumption and metabolic hormone profile in lean and obese men

Principal Investigator(s): Tina Wong

Co-Investigator(s): Dr Vicki Harber (492-1023) and Dr Gordon Bell (492-2018)

Part 2 (to be completed by the research participant):

Do you understand that you have been asked to be in a research study? Yes No

Have you read and received a copy of the attached Information Sheet? Yes No

Do you understand the benefits and risks involved in taking part in this research study? Yes No

Have you had an opportunity to ask questions and discuss this study? Yes No

Do you understand that you are free to refuse to participate, or to withdraw from the study at any time, without consequence, and that your information will be withdrawn at your request?

Yes No

Has the issue of confidentiality been explained to you? Do you understand who will have access to your information?

Yes No

This study was explained to me by: _____

I agree to take part in this study:

(Signature of Research Participant)

(date)

(printed name)

Witness _____
(signature)

(printed name)

I believe that the person signing this form understands what is involved in the study and voluntarily agrees to participate.

(Signature of Investigator or Designee)

(Date)

All participants in research projects are advised that the information they provide, and any other information gathered for research projects, will be protected and used in compliance with Alberta's Freedom of Information and Protection of Privacy Act (FOIPPA).

Subject Information

Name: _____

Date of Birth: _____

Address: _____

Age: _____

Phone # _____

email: _____

Height: _____

Weight: _____

BMI: _____

How many days per week do you exercise? _____

What type of exercise? _____

Group classification:

Please circle yes or no:

Yes	No	Reported history of diabetes, hypertension, heart disease, other?
Yes	No	Do you smoke?
Yes	No	Weight Instability within the last 3 months (change of ≥ 3 kg)?
Yes	No	BMI > 34.9?
Yes	No	Inability to exercise (orthopedic limitations, etc.)?
Yes	No	Medications? (If yes, please list)

RESIDUAL LUNG VOLUME and HYDROSTATIC WEIGHING

Name: _____

Date: _____

Pulmonary Information

Age: _____ Temperature ($^{\circ}\text{C}$): _____ Trials _____

Gender: male Barometric Pressure: _____ RV _____

Race: _____ VC _____

Height: _____ TLC _____

Weight: _____

Hydrostatic Weighing

Body Weight (kg): _____ Temperature ($^{\circ}\text{C}$): _____

GI Tract Volume (liters): males = 0.1150 Dead Weight (kg): _____

Residual Volume: _____ %Fat Formula: Brozek

RV Multiplier: 1.00

Fat Free Mass: _____

% Body Fat

Fat Mass: _____

Body Density: _____

Notes:

MAXIMAL AEROBIC POWER

Name: _____

Date: _____

Height: _____

Age: _____

Weight: _____

TIME	RESISTANCE (KP)	RPM	HR

VO₂max: _____ ml/kg/min _____ l/min

HR @ VT: _____

% VO₂max at VT: _____

PO @ VT: _____

3 Day Dietary Intake Record

For 3 consecutive days, you will record your daily intake of foods and fluids. It is imperative that you record EVERYTHING that you eat and drink (water as well). Be as ACCURATE as possible when you do so!! If in doubt about what to record, write everything down or save the information in case you need to use it later. Below are some hints about improving accuracy when recording dietary intake.

Hints for Recording Dietary Intake:

ACCURACY

1. **Accurate Measurement.** Read the weights or volumes of foods or drinks from packages or wrappers. Example: milk carton, juice box, chocolate bar, potato chips. A "fistful" of meat = 100 gm, "fistful" veggies = 1 cup, 1 cheese single = 1 oz.
2. **Method of cooking** Indicate how your food was cooked. Example: fried, steamed, baked, broiled, BBQ, etc. Record any additional oils or spices were used in the cooking process. When cooking vegetables, record the state of the raw vegetable (canned, raw, frozen) and the method used to cook them. Also, take note of what you add to canned soups: water, milk or nothing. Record what you add and the can size used.
3. **"Extras"** Don't forget the EXTRAS. Example: ketchup, mustard, mayonnaise, gravy, or butter.
4. **Food Types** Be specific about TYPES of food/drink. Dairy products can be tricky. Always record the % milk fat and take note of low fat items. For breads, buns, submarine buns, please record if they are white or brown or any other kind. Eggs come in 3 sizes; small, medium and large; record the one you eat. Fruit juices are labelled "Drink", "Beverage" or "Real Fruit Juice"; provide this information. Also note if they are sweetened, unsweetened or from concentrate. Example: cheddar cheese, 2% milk, margarine or butter. Whenever possible, identify brand names of the foods.
5. **Cooked or Dry Measurement** Indicate whether the food measurement was taken before or after it was cooked. Example: state whether you measured meats, rice or pasta before or after it was cooked. The energy content of 1 cup of uncooked rice is very different from 1 cup of cooked rice !!
6. **Specific Parts** Indicate the exact part of the food you ate or what was removed before eating. Example: chicken (white or dark, bone in or out, skin or skinless), baked potato (skin or skinless), ground beef (lean, extra lean, or regular).
7. **Labels** Read the nutritional information label from the container (box/can/bag). This will help identify specific brand food nutrients. Please bring in the label if possible, otherwise, provide all the information the label contains.
8. **Vitamins, Minerals or other Supplements** You can record the vitamins and minerals you take but these will not be analyzed by the nutritional program. The analysis will be limited to CHO, protein and fat intake. Protein or amino acid supplements will need to be entered using information from the label.

BEVERAGES

9. **TEA AND COFFEE** should be included as well along with the cream, milk and sugar you add.
10. Don't forget **WATER**.
11. Yes, you do have to record **BEER and ALCOHOL** as well.....!!

PREPARED OR RESTAURANT MEALS

12. **Use PORTION PAKS** whenever possible. Example: salad dressing, butter, jams, peanut butter, cheese. It is easier to quantify the volume of these foods...1 portion pak = 1 tablespoon.
13. **Fast Foods.** Include FAST FOOD items by name. Example: McDonald's, Pizza Hut, Wendy's.
14. **Recipes.** Record the amount/volume of each ingredient, the number of servings the entire recipe makes, and how many servings or what volume you ate. Example: recipe makes 30 cookies and I ate 5, or the recipe makes 12 servings of lasagna and I ate 2 servings.
15. **Restaurant Meals** When you eat at a restaurant (other than a fast food place, eg. Earl's), record the name of the meal you ate, list the amount or volume of each ingredient (don't include what you don't eat).

TAKE THE RECORD BOOK WITH YOU AT ALL TIMES.....IT'S EASIER TO RECORD WHAT YOU'RE EATING

MENU ITEM		UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating		Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
E V E N I N G M E A L	Menu Item					
	Toppings or Additives					
	Menu Item					
	Toppings or Additives					
	Menu Item					
	Toppings or Additives					
	Menu Item					
	Toppings or Additives					
	Menu Item					
	Toppings or Additives					
Mark (X) One Category	Eaten at Your Home			Day Three		
	Eaten Away From Your Home					
	Did Not Eat					

MENU ITEM		UNIT OF MEAS.	No. of Units	DESCRIPTION OF MENU ITEM		
Enter all foods, beverages, etc. consumed as menu items. For every menu item, include any toppings or additives added to the menu item at the time of eating		Enter the Word "cup" "ounce" "number" "teaspoon" "tablespoon"		Brand	Type of Flavour	Method of Cooking
M O R N I N G M E A L	Menu Item	eggs	number	3	Donland	scrambled
	Toppings or Additives	ketchup	tablespoon	2		
	Menu Item	sausage links	number	2	Schweizer	fried
	Toppings or Additives					
	Menu Item	whole milk choc mix	cup	2	Silverwood	
	Toppings or Additives		tablespoon	2		
	Menu Item	corn flakes	cup	2	Kellogg	corn flakes
	Toppings or Additives	whole milk sugar	cup	1		
	Menu Item	banana	no.	1		
	Toppings or Additives					
Menu Item	multi vitamin	number	1	Ose-A-Day		
Toppings or Additives						
Mark (X) One Category	Eaten at Your Home		X	Sample Day		
	Eaten Away From Your Home					
	Did Not Eat					

Baseline

Name: _____ Date: _____ Time: _____

(Resting Metabolic Testing)

TIME	HR	TEMPERATURE	BLOOD
------	----	-------------	-------

-30*			(#1)
-5	C a l i b r a t i o n		
0* start V02 collection			(#2)

	02	C02
Ref		
Cal		

(mouthpiece, sitting)

30*			(#3)
-----	--	--	------

50*			(#4)
50-60	C a l C h e c k		

	02	C02
Ref		
Cal		

***** SwitchOver *****

Exercise/EPOC

Name: _____

Date: _____

Time: _____

(V02 Exercise Testing)

TIME	HR	TEMPERATURE	BLOOD
------	----	-------------	-------

-30*			(#1)
-5	C a l i b r a t i o n W a r m - u p		
0* start V02 collection			(#2)

	02	C02
Ref		
Cal		

(mouthpiece, sitting)

30*			(#3)
35 sit with mouthpiece			

50*			(#4)
50-60	C a l C h e c k		

	02	C02
Ref		
Cal		

***** SwitchOver *****

Name: _____

(canopy, lying)

TIME	HR	TEMPERATURE	BLOOD
------	----	-------------	-------

1:10*	*		(#5)
1:25-30	* Cal Check *		(#6)

02	C02
Ref	
Cal	

1:50*	*		(#7)
2:00--:05	* Cal Check *		

02	C02
Ref	
Cal	

2:10*	*		(#8)
2:30	*		(#9)
2:30	* Cal Check *		

02	C02
Ref	
Cal	

Blood Sampling Procedure

TUBE #	EXERCISE TIME	ACTUAL TIME	TIME + 45 MIN	SPIN 10 min @ 2500	Freeze
					-80°C
1	-30 min				
2	0 min				
3	30 min				
4	50 min				
5	1:10				
6	1:30				
7	1:50				
8	2:10				
9	2:30				

Procedure:

1. Get sample from nurse
2. Decant into tube (vacutainer)
 - ❖ very slowly (to avoid bursting RBCs)
 - ❖ Along side of tube
3. Pipet 500 µl into culture tube => vortex and put on ice 5 min => centrifuge 10 min
4. Label time
5. Let clot for 45 min. Spin. Then draw out serum. (hormone blood)

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